THE
LARYNGOSCOPE.

VOL. L MAY, 1940. No. 5

New York, Apr. 15, 1940

To the members of the Editorial Staff of the Laryngoscope:

As the LARYNGOSCOPE is now in its fiftieth volume, and as the Managing Editor, Dr. Max A. Goldstein of St. Louis, has just reached his seventieth anniversary, it is my thought, as the senior in service of the editorial staff, that it would be an appropriate gesture on the part of the collaborators and of the editorial staff to express their comments as to the status of the LARYNGOSCOPE in the field of otolaryngological literature, and any comments they may wish to make in the form of a letter to Dr. Goldstein. These letters will be published in the May 1940 issue of the journal. A photograph of the writer should be sent with his letter, so that same may appear with his comments.

Dr. Goldstein has spent the greater part of his professional life in producing a scientific and dignified journal in the field of Otolaryngology, and the remarks of his associates in appreciation for his worthy efforts should make very interesting reading.

May I ask a prompt response to this request. Thanking you for your interest in this thought, with kind greetings.

Sincerely,

M. D. Lederman

New York, April 22, 1940

Dear Dr. Goldstein:

Having been associated with the LARYNGOSCOPE since its birth in 1896, it is with sincere affection and appreciation that I offer my cordial congratulations and best wishes to the founder and Editor-in-Chief, for the splendid and scientific contribution that he has made to otolaryngological literature. Your personal efforts towards the rehabilitation of the deaf, together with that outstanding institution, The Central Institute for the Deaf, located in St. Louis, which is the result of your indefatigable labors, will be an everlasting monument to your progressive thought.

Your constant desire to aid the underprivileged is thoroughly appreciated by those who are favored by your friendship and experience. Many individuals barred from useful activities, have been restored to active careers due to the successful methods originated and taught under your leadership.

The LARYNGOSCOPE is but another illustration of your progressive, unselfish spirit. Representing the latest thought in Otolaryngology, its pages are open to all who offer any noteworthy contributions.

It has been a great honor to have been associated with you for nearly half a century, and it is with keen appreciation that I extend my personal congratulations upon your seventieth anniversary and sincerely trust that you will be spared to the community and the profession for many more years. Sincerely,

M. D. Lederman

Philadelphia, Pa., April 24, 1940

Dear Doctor Goldstein:

Please accept congratulations on your achievements.

It is a great distinction to have achieved the high place you occupy among the otolaryngologists of the world, and especially so to have done this notwithstanding a tremendous load of clinical and literary work. In the second place you are to be congratulated on having founded one of the great otolaryngologic magazines in the world's literature. I well remember the hard struggle of the early days in laying the foundation for this magazine.

You deserve happiness and the contentment that comes with great achievement, and I hope you will have both.

Yours sincerely,

Chevalier Jackson

Chevalier Jackson

New York, N. Y. May 3, 1940

Dear Dr. Goldstein:

Staff editorship on The Laryngoscope is considered by me a very high honor. Fifty Volumes of the Laryngoscope have been published to the betterment of all those engaged in the study and practice of otolaryngology and the high standards of this monthly publication have been improved and maintained under your long editorship.

It is also with pleasure that I note that you are celebrating your seventieth birthday Anniversary. Please accept my felicitations and best wishes and many, many happy returns of the day.

With highest personal regards and esteem--

Yours sincerely,

C. J. Imperatori

Charles J. Imperatori

Philadelphia, Pa., April 23, 1940

My dear Dr. Goldstein: -

It gives me great pleasure to add my voice to the many expressions of appreciation to you, dear friend, at this 50th Anniversary time. It cannot fail to give you great joy to have accomplished so much and rendered such service to our specialty.

It was my pleasure to see and indelibly record in memory at the Silver Jubilee the results of your work as Director and Founder of Central Institute for the Deaf. Your contributions and fundamental research in defective speech and speech training, and above all, your genial cooperation with every effort which is made for progress, particularly in otology, have won our complete gratitude.

We congratulate you on this long and successful period of service and feel that it is still a beginning but not a conclusion of your great work. It has been a particular pleasure to me to have each month these fine referat reviews of important special topics assigned to men most interested in them.

Again expressing my sincere congratulations to you and the LARYNGOSCOPE, who are, after all, almost one and the same, I remain,

Very sincerely,

James A. Babbitt

Los Angeles, April 14, 1940

Dear Dr. Goldstein:

As an associate editor to the Managing Editor and Publisher, may I call your attention to the tremendous number of adjectives in our language?

There are the good and the bad, the attractive and the unattractive. For many years I have known that the "good" ones apply to you -- but I can now tell you, because you have become of age:

"Generous, scholarly, considerate, courageous, constructive, creative, tireless," -- and I was about to add "truthful," but I don't believe it, when you pretend you are 70 years old!

With affection through the years--

Isaac H. Jones

Boston, Mass., April 26, 1940

Dear Dr. Goldstein:

I have it on reliable authority that you have just attained the scriptural three score and ten years. May I at this time offer my sincere congratulations and hope that the specified strength for attaining fourscore will surely be available.

The retrospective view of your many achievements must be most gratifying. One of my earliest recollections of medicine is that of a pile of orange colored LARYNGOSCOPES on my father's desk, doubtless unread but gradually to be perused as time was available. My own more recent adventures in the editorial role afford me profound appreciation of the tasks which you have accomplished in the fathering of the journal. Medical and surgical proficiency have never been a guarantee of literary ability and the constructional and grammatical devastation often committed by your contributors must have been a sore trial to you. It is to be hoped that those of us whom you have chosen for editorial work may be able to lighten this burden and to still further enhance the value and reputation of the LARYNGOSCOPE.

I regret only that my father, to whom you were one of the closest of friends, is not here to join me in the sincerest of best wishes at this occasion.

Very cordially yours,

Lyman Richards

Lyman Richards

St. Louis, Mo., April 23, 1940

Dear Dr. Goldstein:

It is indeed a pleasure to have this opportunity to congratulate you, not only for your excellent contributions to otolaryngology, but also for the splendid manner in which you have edited the LARYNGOSCOPE through fifty volumes of publication. We all appreciate the tireless efforts you have made to give the field of otolaryngology the benefit of your ability always to separate the "wheat from the chaff." The LARYNGOSCOPE has, therefore, supplied the Vitamin "E", the fertility, which is so necessary for the growth of our knowledge of otolaryngology.

With best wishes for your continued activity as one of the "Generals" in Otolaryngology, I am,

Sincerely yours,

French K. Hansel

French K. Hansel

Worcester, Mass., April 30, 1940

My dear Dr. Goldstein: -

Word comes that the LARYNGOSCOPE reaches its fiftieth volume and its managing editor and chief factotum nears his seventieth birthday, all at the same time. What a period for rejoicing this is! You now look back over the quickly passing years, each so full of adventure, and can say: "it was well done. I have run my course and have kept the faith. Now for the still more exciting years of opportunity and service that are ahead!"

Dignified in its full maturity, and proud in its record of accomplishment and service, the LARYNGOSCOPE reaches the half-century mark and looks forward with zest to the eventful and stimulating years that are to come. Permit me to join with its host of friends in hearty congratulations to the efficient and conscientious staff, and their dynamic leader.

I am honored that at this juncture in your busy life, you have seen fit to stop just long enough to take me into your official family where I may share in your rich experience and seasoned wisdom.

Best wishes, Chief!

Sincerely Yours,

Gordon Berry

Gordon Berry

Chicago, Ill., April 23, 1940

Dear Dr. Goldstein:

On the occasion of the Fiftieth volume of the LARYNGOSCOPE and the Seventieth Birthday of its creator, I wish to express my sincere congratulations.

The LARYNGOSCOPE which is known all over the world is your own work and today you can enjoy the proud feeling that your many efforts led to a great success. I am sure that the success is going to persist and even to increase, not only to embellish the Seventieth Birthday of its founder but also to spread and to enlarge the glory of American Otolaryngology.

Very sincerely yours,

Hans Brunner

St. Louis, Mo., May 2, 1940

Dear Doctor Goldstein:

Please accept my sincere felicitations upon your having accomplished the allotted span of time and upon the fiftieth anniversary of the LARYNGOSCOPE.

Fifty volumes of uninterrupted publication under one editor-manager is, so far as I know, a unique record for any professional journal and one to which you may look with pardonable pride. During this entire period the LARYNGOSCOPE has occupied a position of dignity and distinction among medical journals, not only in America, but throughout the world. Little wonder that it has been chosen as the official organ of publication by so many distinguished oto-laryngologic organizations.

When I come to consider your life, I am amazed that one individual could achieve distinction in so many fields of activity, even though they be more or less related: distinguished otologist, founder and editor of the LARYNGOSCOPE, founder and director of the Central Institute for the Deaf, the Institution responsible for training and teaching methods that have enabled thousands of children handicapped by deafness to establish normal contact with the world and become useful and happy members of society; teacher, scientific investigator and organizer. What a galaxy of achievements for one man.

Sir, I salute you upon this occasion and wish for you the long life and continued health that will permit you to see the full fruition, not only of all of your previous efforts, but of the even more splendid plans which I know that you have for the future.

Yours very sincerely,

Arthur M. Alden

Arthur M. Alden

St. Louis, May 4, 1940

Dear Dr. Goldstein:

It is an honor and a privilege to join the editorial staff of THE LARYNGOSCOPE with you, especially at this time when your shining light is ever increasing and the field of your influence is being so well recognized. Your enthusiasm and work will always be a goal and objective for the Editorial Staff.

Most sincerely,

J. Barrett Brown

J. Barrett Brown.

Boulder, Colo., April 23, 1940

Dear Dr. Goldstein:

Congratulations and best wishes for many more useful years. It must afford you a great deal of well deserved pride and satisfaction, at three score and ten, to see the LARYNGOSCOPE marked volume fifty this year.

There are in the United States some seven or eight thousand otolaryngologists practicing our specialty. Not all of these write articles for medical journals, but of all those who write only a few are privileged to be managing editors and publishers of a journal. You have by your steadfast and intelligent effort, guided by a thorough knowledge of otolaryngology, placed the LARYNGOSCOPE among the leading ear, nose and throat journals of the world. Not all great men live to see the mature results of a long and scientific life, but you now enjoy this unique distinction.

I am only one of many friends in this country and abroad who join in wishing you many more happy, useful years. Yours very sincerely,

Frank R. Spencer

New Orleans, La., April 25, 1940

Dear Dr. Goldstein:

All praise and warmest greetings to you on the celebration of your own septuagenary and the semicentennial of your but slightly less renowned child, the LARYNGOSCOPE!

Though the lush fruits of your labor have fallen generously in many directions, the LARYNGOSCOPE is certainly not one of the least; and being one of the first, it cannot but be very dear to your heart. Yet, if you esteem it for that reason, your glow of satisfaction is as justifiable for many another.

In its fifty volumes, the LARYNGOSCOPE has published some of the truly monumental pieces of otolaryngological literature. Both at home and abroad it has been widely respected and genuinely admired for the quality of its style and content; and it is but fitting that it has been selected as the official organ of some of the most distinquished otolaryngological societies of America.

To you, both for good work and its accomplishment, Salute! Cordially,

Francis E. LeJeune

Toronto, Canada, April 25, 1940

Dear Doctor Goldstein:

I wish to send you my heartiest congratulations on reaching your seventieth anniversary. That birthday is a milestone to any man. I am happy to believe that you have reached this in a state of physical and mental well-being that is granted to but few.

I hope that you will have many happy years before you.

It is likely that you will have these for you have the gratification of having a host of loyal friends and many admirers who are proud of you and the work that you have done for Otolaryngology. The praise and good-will of such would be enough but you have much more than this for you have the great inward satisfaction of knowing that you have run your race well, struggled hard over the difficulties, have surmounted these and have reached the goal you have worked for. I am sure that you have nothing but proud and happy memories.

I look upon you as a very worthy friend of my father, Dr. David James Gibb Wishart, of whose memory and achievements I am very proud. The picture he painted of your characteristics time has shown to be just, fair and true and of these I need say no more than that I too am proud to have your friendship.

It must be a pleasure to you to see that the LARYNGOSCOPE has reached its fiftieth volume and yet gives an appearance of a better quality than at any stage in its past. This should augur well for its future.

Please allow me to express my sincerest good wishes for many happy years to be.

Yours very truly,

D. E. S. Wishart

Newark, New Jersey, May 4, 1940.

Dear Doctor Goldstein:

My heartiest congratulations to you in the celebration of your seventieth anniversary! As you look back over the span of three score and ten years, it must be with a very deep feeling of having accomplished that which is so worthwhile in life to know that 'The Larynogoscope' - an international Monthly Journal Devoted to Diseases of the Ear, Nose and Throat, which you founded in 1896, and to which you have devoted so many years of untiring, unselfish endeavour, is now in its 50th volume. Under your expert guidance 'The Laryngoscope' is one of the outstanding journals in the field of otolaryngology. This has all been due to your far-sightedness and minute attention to every detail.

I feel that it is a real privilege and indeed a pleasure to be associated with you as a member of 'The Laryngoscope' Editorial Staff, and it is my sincere wish that you will continue as Managing Editor and Publisher for many more years to come.

Very sincerely yours,

Henry B. Orton.

Los Angeles, April 23, 1940

My dear Dr. Goldstein:

Upon this double occasion of the celebration of your seventieth birthday anniversary and your presentation of the fiftieth volume of the LARYNGOSCOPE, I extend my warmest felicitations and heartiest congratulations.

Seldom does there come to any one the privilege of serving his fellow-man in so many varied capacities and with such outstanding merit as has fallen to your lot. As one of few great pioneers in your chosen specialty, you have been instrumental in elevating the standards of otolaryngologic research and practice to a high plane; in your capacity as teacher there have passed thru your hands legions of men and women whose work shall stand forever as a monument to your own momentous scientific contributions. You have been singularly honored by your fellows thru appointment to many high offices, each of which you have occupied with distinction, to the credit of our profession and with limitless glory to yourself. As an organizer of things worth while, few can point to so many distinguished achievements. Not the least of these is the LARYNGOSCOPE which thru nearly fifty years has been perpetuated, and has grown to occupy its present constantly increasing sphere of usefulness, solely because of your own personal enthusiasm, vigor and ability.

Above all however, there have come to your door from far and near countless, pleading thousands, destined to travel the road of life carrying in hopeless servitude the burden of an infirmity from which there seemed no relief. To each of these you gave freely of your genius, sending them forth anew, stalwart and determined to meet life's problems with happy hearts and sound bodies, rehabilitated - almost reborn! In the sincerity and depth of their gratitude, and in the admiration of all who saw, is carved indelibly, and far more effectively than anything we can do, the tribute we seek to pay you as leader, healer, friend and teacher.

For these reasons, and for many others, we salute you!

With thanks to God that you have been spared thru these seventy fruitful years, and with prayers for your continued health and happiness, I extend my sincere congratulations, admiration and respects.

Yours most cordially,

Joel J. Pressman

Joel J. Pressman

London, May 10, 1940.

Dear Dr. Goldstein:

In the last decade of last century a young and struggling practitioner in London had just cast off the chrysalis case of general practice and was endeavouring to stretch his wings in the world of Oto-Laryngology. In the early days of their first, fluttering efforts he was cheered and flattered by receiving from an unknown colleague in St. Louis a polite and cordial invitation to act as European correspondent to "The Laryngoscope", - a monthly journal then in its second year. He was further encouraged by being invited to contribute a communication and was honoured by finding it printed on the first page of Vol. 4 in 1898.

This early contact was the start of a friendship of 42 years, during which I have steadily admired the courage, devotion and skill with which you have conducted this Journal of international repute. In those days, I was eking out a precarious existence by sub-editing a monthly journal of general medicine, - "The Practitioner". As a certain Lord Salisbury, - a journalist in early life, who afterwards became Prime Minister of England, - had said "Journalism was a good training and career for the young man of literary tastes, - if he knew when to give it up." I felt obliged to do this; but you, being possessed of more vigor and altruism, have continued to edit during 44 years the magazine you founded. Besides this you have done many other excellent things, - scientific, professional and social. Your study of the problems of the deaf, and your efforts for their relief, have been truly philanthropic. The good status of your Journal has secured the support of scientific societies and attracted a distinguished list of contributors. Your love of art and general culture has, I have no doubt, comforted you with wide and generous interests.

I am gratified and well pleased at being honoured by having my name associated with your work and with your band of brothers and co-workers.

On this happy anniversary I send you my warm greetings and kind thoughts; in the words of that good fellow, Shakespeare,

"Health and fair time of day,
Joy and good wishes,"

and I remain, always,

Yours sincerely,

St. Clair Thomson

St. Clair Thomson

PROGRESSIVE DEAFNESS, OTOSCLEROSIS AND CLOSELY RELATED SUBJECTS.

AN ABSTRACT OF THE AVAILABLE LITERATURE PUBLISHED DURING THE YEAR 1939.

DR. JAMES A. BABBITT, Philadelphia.

After a careful review of more than 100 contributions, with the valuable assistance of my associate, Dr. Louis E. Silcox, it appeared that the relation of progressive deafness to otosclerosis was still unsolved, although nearer to solution than before. Consequently, the title of this review will still embrace a number of conditions which belong to a closely related group. An effort was made to collaborate opinions in our important otologic centres and obtain some correlated group opinion, at least in definition. Reports from different research centres throughout the country, however, were so varied that no satisfactory composite definition could be obtained and consequently no adequate measure of differential diagnosis. In the approach to agreement above noted, progress would seem to have been made in a more satisfactory isolation of otosclerosis in the progressive field. Perhaps this is largely due to a widening of the gap between the conductive deafness of simple tubotympanic involvement and otosclerosis with distinctive osseous change, and, on the other hand, increasing the margin between the progressive deafness combined with a true osteoporosis and that of intrinsic cochlear pathology.

In the articles abstracted, the authors' opinions will be reported without comment as to their views on this pathologic entity. As they seem to follow rather classic groups, for convenience in treatment these might be outlined as follows:

Group 1: Clinical Studies and Research.

Group 2: Medical Therapeusis and Systemic Readjustment.

Group 3: Mechanical and Surgical Procedures.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 1, 1940.

GROUP 1: CLINICAL STUDIES AND RESEARCH.

Nager[1] has presented another of his definitive reviews, under the title, "Clinical and Pathological Anatomy of Otosclerosis." He stated that otosclerosis was recognized as a localized disease of the labyrinthine capsule and stapes base, with primary bony tissue progressively undermined and riddled. The process frequently encroached upon the annular ligament and might even penetrate the labyrinth.

Otosclerosis has been observed among all civilized peoples, whether its origin is hereditary or dates from some extinct human race is not yet known. Its frequency in individual areas has been difficult to determine because of its latent localization in some functionless insignificant part of the labyrinthine capsule.

The author established otosclerosis in 20 per cent of 1,000 cases of deafness examined, and quoted Shambaugh as having 30 per cent in similar research. Insufficient data as to the macroscopic and microscopic conditions in which otosclerosis is invariably associated with deafness have made these findings only conditional. Bezold held that otosclerosis was always present in young individuals suffering from progressive bilateral deafness.

The exact beginning of the disease is difficult to determine because of the gradual onset with loss of hearing. In 1,146 cases studied by the author, it was found that the disease began in 50 per cent between the ages of 16 and 30 years, with somewhat higher frequency between 21 and 25 years. In only 13.5 per cent of the patients observed was onset noted prior to the sixteenth year, and in only 14 per cent after the age of 25 years. Ten per cent were affected in only one ear at first, the other remaining normal for a period of years. The onset was slightly earlier in unmarried individuals and the course was generally progressive.

Clinical manifestations in otosclerosis included auditory murmurs and vertigo. In 1,146 cases examined, 773, or 67.3 per cent, had subjective murmurs of varying intensity. Symptoms of vertigo appeared in 327. Vestibular lesions could not be established. The tympanic membrane picture was normal in 802 cases. In well established otosclerotic cases, exostosis

formation in the external auditory meatus was found. Paracusis Willisii was reported in about 20 per cent of the cases.

From the pathologic standpoint, the fact of a more or less localized disease of the labyrinthine capsule, uni- or multifocal, gradually extending from predilection sites to other parts, has aided in the study.

Slightly different phases of the process were:

1. Osteoblastic resorption of the old bony tissue, with fibrous osteoblastic bony tendency.

2. Substitution of scrofulous bone (noted by means of hematoxylin eosin blue).

3. Alteration and substitution of tissue by fibrile bone.

4. Development of layers of scrofulous bony tissue.

5. Further infiltration of lamellar bone persisting to sclerotic bony area with bone marrow.

Lurie[2] carefully discussed the question, "What Is Perception Deafness from a Physiological and Histological Basis?"

Hearing did not concern the ear alone. What one heard was the interpretation by the brain of nerve messages or impulses sent to it by the sense organ of sound (organ of Corti). This interpretation depended upon the mental capacity of the human or animal.

Two phases of deafness or the inability to hear were presented:

1. Inability for sounds to reach the sense organ — conduction deafness.

2. Inability of the end-organ or brain to receive or interpret the sound waves — perception or nerve deafness.

Perception deafness resulted from the inability of an individual to interpret the sounds heard in intelligent language. The neuroepithelium of the perception organ is the organ of Corti.

The tectorial membrane should be regarded as a modified otolithic membrane, with functions similar to the otolithic membrane of the vestibular apparatus. It is a structure

which aided in the movement or bending of the hairs in the external and internal hair cells.

The organ of Corti was essentially an organ of touch and had developed from the embryonic skin. The tectorial membrane was in ultimate contact with the organ of Corti.

The stria vascularis, similar to the choroid plexus of the brain, secreted the endolymph.

The basilar membrane on which the organ of Corti rested had been the center of attack since Helmholtz first developed the harp theory of resonance on the basis of difference in width of the basilar membrane.

Was the vibration of the basilar membrane necessary for hearing? The organ of Corti rested partially upon the basilar membrane. The external hair cells and external pillar cell rested on membrane, and the internal hair cells and internal pillar cell on bone, which allowed greater movement for the former. The external hair cells, upon a movable membrane, necessarily received greater stimulation. Long-continued vibration caused changes in the external before the internal cells. Accompanying this degeneration of external hair cells, the loss of response from the organ of Corti by the Wever and Bray method was from 10 to 40 dcb. This degeneration of external hair cells accompanied loss of hearing typical of boilermaker's deafness.

The external hair cells responded to stimuli of weak nature, the internal to those of greater intensity. Animals deafened by the use of quinine showed degeneration of the external hair cells.

Perception deafness in greater or lesser degree could be considered as a degeneration of the external hair cells in the organ of Corti. A scattered degeneration caused slight losses; a more general degeneration, greater losses in hearing. A complete loss occurred with degeneration of both external and internal hair cells.

Nerve supply of the organ of Corti differentiated the function in these two types of cells. A single nerve innervated one or two internal hair cells — the nerve to the external hair cells were in contact with a large number. There was no fine discrimination of pitch by the external hair cells.

The cochlear nerve was a bipolar sensory nerve similar to any other postganglionic nerve. The organ of Corti could remain functioning long after the cochlear nerve had been damaged, which might explain the recovery of hearing in cases that have had complete deafness during the course of the disease. Here the lesion was of the nerve itself and when regeneration occurred and neuritis disappeared, hearing recovered because normal connections were re-established between the organ of Corti and the central nervous system. The great mystery was how we heard a variety of sounds at one time and kept them distinct and separate.

Crowe and Baylor,[3] following the work on impaired hearing for high tones by Crowe and Guild in 1938, presented an exhaustive paper on "The Prevention of Deafness" in 1939. This outstanding paper belongs in the realm of pure research as well as practical therapeutics.

The work which had been done at Johns Hopkins University since 1934 in the accumulation of some 15,000 records of accurate hearing tests was reviewed. As the number of audiometric records was increased, it became apparent that impaired hearing for high tones was extremely common in children, as well as in adults. Although the classic teaching in otology had been that impaired hearing for high tones and good hearing for low tones indicated an inner ear or nerve lesion, the frequency with which this disorder was found led to doubt as to the correctness of that conclusion. This doubt was maintained after the observation was made that some children regained their hearing of high tones after the removal of enlarged tonsils and adenoids. The fact that hearing did not always improve after operation led to examination of the upper respiratory passages which required the nasopharyngoscope, sometimes under general anesthetic. In these studies they found the compensatory development of lymphoidal tissue nodules on the lateral and posterior walls of the pharynx, called granular pharyngitis, and, in addition, overgrowth of lymphoidal tissue in and around the pharyngeal orifices of the Eustachian tube. This abnormal growth of lymphoidal tissue partially obstructed the tube with hypersecretion of mucus and chronic irritation in the tubes and middle ears. The earliest symptom of such partial obstruction was impaired hearing in the tones between 10,000 and

16,000 d.v. Good hearing in the middle of the scale, from 250 to 3,000 d.v., masked the insidious changes in the higher tones. The work of Heinecke at the University of Leipzig, showing that lymphoidal tissue was more susceptible to irradiation than the adjacent epithelium, muscle and bone, suggested the use of radium and Roentgen rays in the treatment of this condition. With the aid and advice of Dr. Burnam, the authors began 10 years ago to treat this tissue with Roentgen rays and radium in an applicator small enough to pass along the floor of the nose. The nasopharyngoscope in the opposite side of the nose kept the application under visual control. The symptomatology of long-continued partial obstruction in the tubal area during childhood was:

1. A slowly progressing deafness that began with the highest tones; pain, tinnitus, dizziness or other symptoms usually absent.

2. Recurring attacks of otitis media in children who have a tendency to lymphoidal hyperplasia.

3. Long-continued discharge in the ear after paracentesis or after simple or radical mastoid operation.

The authors carefully described the use of radium and Roentgen therapy. Five cases were reported, with appropriate audiogram illustrations. The authors believed that if the causal condition was recognized and properly treated before the age of 15 years hearing usually returned to somewhere near the normal level; after that age, the results were far less satisfactory. After the age of 15 years, secondary changes in the middle ear might be advanced beyond repair by any treatment.

Irradiation did not permanently remove hyperplastic lymphoidal tissue, but relatively small doses, without injury to pituitary, nasopharynx or inner ear, kept this in abeyance during the age period in which it grows most actively.

It was felt that if school children in the primary grades were examined by the nasopharyngoscope at least once a year, and the hyperplastic lymphoid tissue around the Eustachian orifice was treated with irradiation as often as was necessary to restore normal tubal function, the number of deaf adults in the next generation could be reduced by 50 per cent.

"The Eustachian Tube. Abnormal Patency and Normal Physiologic State" was discussed by Perlman.[4] The author discussed the method for quantitative determination of the patency of the Eustachian tube by the use of the manometer of the common blood pressure apparatus, together with observation of the drumhead during Valsalva and Toynbee's maneuvers, which permitted an easy determination of the function of the Eustachian tube. He maintained that unless quantitative studies were made, the pathologic patent Eustachian tube was likely to be overlooked.

When the tube opened to pressures below 20 mm. of mercury, it might be considered pathologically patent. When extreme patency existed, even the small pressures developed in respiration and phonation were sufficient to open the tube. The author then enumerated many factors which affected the patency of the Eustachian tube and factors which caused a pathologically patent tube to remain open.

The study of a group of cases here presented indicated that a lack of tonus of the muscles which affect the Eustachian tube and were supplied by the Vth nerve were the principal factors in producing the clinical entity.

Bunch,[5] in discussion of the "Progress of Deafness in Clinical Otosclerosis," noted the general agreements as to the progressive nature of the deafness in cases of clinical otosclerosis, its progress depending on the individual, and cited Lederer, Harrison and Holmgren in agreement with this. The audiometer was the only test of value in estimating the progress of an otosclerotic's hearing, whether in observing both operative effects or following the clinical course of the disease. The author emphasized the psychic element, influence of fatigue in such deafness, and the importance of their estimate in the analysis of an otosclerotic patient and judging estimate of his progress.

The following twofold solution for this problem was presented: First, testing the hearing of many patients throughout life and carefully noting changes; second, testing a large number of cases at different age levels and statistically determine the amount of change at each level. In no case was the diagnosis to be made simply by audiometer alone; to that should be added tuning fork tests and notation of the absence

of tubal and tympanic pathology. The author quoted Suggit in the statement that no treatment of otosclerosis had value without sustained improvement of appreciably more than 10 dcb. over the greater part of the 64 to 8,192 register. The author's study had been limited to six years, so no comprehensive conclusions could as yet be made.

In some cases there had been no measurable increase in hearing loss. Where deafness had been increased, no obvious factor in the patient's health nor any peculiar result in functional tests had indicated whether or not change in hearing might be predicted. The author concluded that time alone was not the factor in determining the progress of the deafness.

"Auditory Murmurs — Their Origin and Treatment" was presented by Meyer.[6] The author believed that auditory murmurs and tinnitus aurium originated within the ear. His hypothetical explanations were:

A. That murmurs arising in the middle ear were conditioned by two factors:

1. Incidents from illness, such as hyperemia, exudate, etc.

2. The acoustic insulation by closing of the ear against external sound impressions, which in healthy individuals drowned out the vascular and muscular murmurs.

(In the middle ear deep-toned murmurs predominated.)

B. That the vast majority of auditory murmurs occurring in the inner ear were due in part to vascular murmurs in the labyrinthine capsule, and in part to otosclerotic structural incidents.

The source of these murmurs was undoubtedly intracellular and should be sought in the sensory cells of the cochlea or in the cochlea proper. As in labyrinth disease there was usually disturbance of hearing for high tones, due to degeneration of a segment of the cochlea, it was natural that through intracellular degenerative processes, high auditory murmurs would again occur.

Tinnitus aurium occurred more frequently and with greater regularity in otosclerosis than in any other disease associated

with deafness. Therefore, otosclerosis should be suspected in cases of middle-aged deafness with tinnitus and normal tympanic membrane findings. Tinnitus aurium was seldom encountered in hereditary deafness and was usually absent in occupational diseases. In central diseases, as in small pontine angle brain tumors, subjective auditory murmurs often occurred as early symptoms.

The origin and severity of tinnitus aurium depended upon so many external factors that all influences must be given special consideration. The doctor stressed moderation in all foods rather than special diet; stated that in general therapy he considered bromide in connection with calcium very effective; and that atropine and belladonna preparations had long been used with success. As vascular disturbance played an important rôle, he found that treatment with caffeine and theobromin in combination with a sedative such as theominal, gave good results.

Voss[7] contrasted "Old and New Views on Otosclerosis," citing the fact that the name, hardening or thickening of the tympanic membrane, was hardly a consistent term, in our present view. Histologic research revealed that in otosclerosis there occurred a change in individual parts of the bony capsule — in place of compact bone, spongy, hollow spaces. This did not represent simple transition, but new, surplus, projection material, extending over the old in tumor fashion. A predilection site was in the bony structures in the region of the oval window; if connected with the base of the stapes, ankylosis occurred with considerable sound conduction disturbance so that sound could no longer be transmitted to the labyrinth. Otosclerotic areas could also arise in other parts of the labyrinth capsule, *e.g.*, the cochlea, round window, and along the auditory nerves, with or without involvement of the stapes base. It had not been proved that the labyrinth affection was directly connected with bone disease; however, occurrence of inner ear disease on the one side, and typical stapes ankylosis on the other, strongly suggested otosclerotic origin. Vestibular symptoms, vertigo, nausea, etc., were the result of either inner ear disease or due to vasomotor conditions. In rare cases of otosclerosis, fistula symptoms were found which were attributed to increased air density and rarefaction in the external auditory meatus.

Instances were cited of improved hearing in noisy places, subways or near a water fall. One author stated that the cause of paracusis was not incidental to sound conduction apparatus but to increased auditory sensitivity to noise. In otosclerosis a deficiency of cerumen in the external auditory meatus was usually found. This was peculiar to older individuals and was due to hypercholesterol anemia.

The tympanic membrane in otosclerotics was usually markedly tender and transparent, and in research a diminution of calcium content in the blood could be established in 80 per cent of the cases. The matter of hereditary transmission has baffled therapeutic research. Most authors agree that prophylactic measures should be taken in preventing the marriage of otosclerotics, or at least the avoidance of propagation and further transmission.

In closing this article, the author reviewed a number of quite variant methods of treatment and made the suggestion that otosclerotics improved in high altitude and did badly in lower, sea air.

Pohlman[8] discussed the "Restoring Forces in the Mechanics of Audition." Audition could be separated into a physical and physiologic component. The physical included all displacements from the sound source to the activation of auditory cells where the physiological component began — to terminate in a central nervous system. The author considered that auditory cells were nonspecific in response.

An excellent diagnosis between conduction and perception deafness was established before the close of the sixteenth century. The Eustachian catheter was used in 1741. Incision of the drumhead was described in 1802. The most outstanding fact regarding auditory apparatus was establishment of the labyrinth liquid in 1774. Weber and Helmholtz were responsible for the generally accepted interpretation of the mechanics of audition today.

The author discussed what he termed the acoustic insulation of the labyrinth, and the theory of the function of the cochlea as a mechanical harmonic analyzer, which might explain an extrinsic activation of the nonspecific auditory cells. He then elaborately described the mechanics whereby the entire system from drum membrane to round window

swung as one unit from the pressure of the applied sound pulse. Next, the restoring forces in the system required the entire unit to swing back to follow the varied displacement in the sound vibrations heard.

That explanation was offered by Weber as producing the transverse vibration to the basilar membrane, which von Helmholtz indicated did not solve the problem of the frequency analysis. The author also discussed recent cases reported by Crowe and Polvogt, also by Alexander, with a dehiscence in the bony septum between the middle and apical turns in six out of 17 cochlea, called the Scala Communis Cochlea. According to the author, "the restoring forces of the auditory system could be located in several places — the drum membrane, the trapped air of the middle ear, the intrinsic muscles, the ligamentus attachment of the ossicles, the elastic annular ligament of the stapes and the elastic tissue of the round window membrane.

According to the author, every case of progressive conductive deafness would reach the stage when, by these venting factors, the amount of vibrational thrust through the stapes would exactly balance that through the round window. The author noted the fact that there have been a number of ventilation operations performed on the labyrinth to improve the hearing. Had these otologists clearly understood the physics of the accepted interpretation of auditory mechanics, they would never have attempted the operation. The only restoring forces in the auditory apparatus were those slowly acting ones which could not respond to sound vibration.

"Studies of Pupils of the Pennsylvania School for the Deaf" were carefully presented by Hughson, Ciocco and Palmer.[9] By way of introduction, the authors discussed the general problem of schools for deafened children and the importance of accurate studies of the deafness status. In this study, the hearing of 487 children was examined for air and bone conduction. This group formed 92 per cent of the children enrolled in the Pennsylvania School for the Deaf. After discarding unreliable responses, the study was comprised of 460 air conduction audiograms and 454 bone conduction audiograms. It was observed that for both the right and left ears, and for both air and bone conduction, the average threshold increased as one proceeded from the low to the high auditory

frequencies. At least 95 per cent could perceive at least one frequency by air conduction in one or the other ear; and 85 per cent perceived at least one in both ears. Total impairment was infrequent. This should stimulate further research in therapy and rehabilitation of these children.

In 227 children acquiring difficulty in hearing after birth, the causes appeared to be as follows: Meningitis, 17 per cent; head injury, 14 per cent; measles, 9 per cent; otitis media, 6 per cent; and scarlet fever, 6 per cent. One and six-tenths per cent of the children had practically normal bone conduction.

The writers felt that in future, reported analysis of hearing should be correlated with clinical examination, family history, social adjustment, scholastic achievement and aptitude for different forms of vocational training. In the majority of children, deafness was bilateral; sex was not a significant factor. Fifty-four per cent of the children were said to have been born deaf. A significant number of children were found with sufficient residual hearing to warrant effort in therapeutic relief.

Ballenger,[10] in a lecture discussion of "Types of Deafness," recognized three general types — conductive, perceptive and progressive.

Conductive deafness was due to lesions in the conductive mechanism and was never a complete deafness. He discussed lesions of the external and middle ear.

Perceptive or nerve deafness, the most profound type of deafness, was due to a lesion in the perceptive mechanism — cochlear, acoustic nerve or brain centre.

Progressive deafness, the third type, was otosclerotic deafness or otoporosis, which might simulate either the conductive or perceptive deafness. He discussed the pathology of this type.

Deafness might also be classed upon etiology. Shambaugh reported that of 5,000 profoundly deaf children in Chicago, 60 per cent were congenital and 40 per cent acquired. The author outlined the following etiology in perceptive deafness:

1. Trauma with fracture of temporal bone, causing unilateral deafness.

2. Drugs, such as quinine, arsenic, salicylates, mercury, alcohol and nicotine.

3. Toxins from infections (mumps, etc.).

4. Various bleeding diseases causing labyrinthine hemorrhage.

5. Severe anemias.

6. Senile type due to arteriosclerosis.

7. Boilermakers and other occupational noises.

"Experimental Production of Deafness in Young Animals by Diet" was reported by Mellanby[11] after a very thorough and extensive piece of work on the production of changes in the labyrinthine capsule and the VIIIth nerve.

He noted the following histologic changes in the labyrinthine capsules of young dogs fed for some months on diets deficient in vitamin A:

1. Degeneration of different degrees up to complete disappearance of the cochlear nerve, the cells of the spiral ganglion and their central and peripheral branches.

2. Degeneration of a lesser degree of the vestibular division of the VIIIth nerve.

3. Overgrowth of bone in the modiolus and of the periosteal layer of the capsule near the brain.

Pressure from this overgrowth of bone apparently caused degenerative nerve changes.

Serous labyrinthitis also appeared in the cochlea of dogs on the vitamin A-deficient diet. This seemed to produce degeneration of the sensory epithelium of the labyrinth, including the organ of Corti and the ampullae of the semicircular canals. Substitution of potato for the cereal element in the vitamin A-deficient diets greatly reduced the abnormal changes in the labyrinth. The base of the skull showed bony overgrowth and deformity in these vitamin A-deficient dogs, which probably caused degenerative changes in other cranial nerves — optic and trigeminal.

Ulrich[12] presented an interesting "History of Deafness in a Family and Its Descendants, Dating from the First Half of the Eighteenth Century." The enforcement of more rigid sterilization laws in Europe has given the problem of deafness a prominent place in otologic research. It was interesting to note that the first laws of this kind were enacted not in a large country but in a small, liberal Swiss canton. The author paid tribute to Albrecht for his differentiation of the various types of hereditary deafness, from which evolved the neuropathic acoustic theory of Hammerschlag. The question of blending of different genotypes in deafness and the contribution of otosclerosis has still been answered only hypothetically.

An intensive family history review was made over a period of five generations. In the beginning of the eighteenth century, a married couple, of Zurich, had perfectly normal hearing. Among their children, three daughters were afflicted with congenital deafness. The marriage of one to a young man of normal hearing was forbidden but afterward consummated. Several of their offspring had normal hearing but there were also three congenitally deaf daughters. Thus, in two succeeding generations six deaf children were born, with only one deaf parent, and actually originated from the union of two people with normal hearing.

Research revealed 390 known descendants of this family but no case of deafness could be ascertained. The author discussed the endogamous and exogamous phases of marriage during 200 years, which would make the reappearance of deafness fairly improbable but by no means precluded its possibility.

It was suggested that if this type of investigation were pursued for a longer period and substantiated, one could be reasonably sure of further disappearance of deafness of the hereditary type, and there would be justification for annulment of strict sterilization laws.

"The Process of Healing in Injuries to the Capsule of the Labyrinth" was discussed by Perlman.[13] A small perforating injury to the capsule of the labyrinth was sealed by a blood clot in a few hours, and swelling of adjacent periosteal layer observed after the first day; fibrous tissue from the perios-

teum invaded the blood clot and replaced it in three days, when a differentiation of the periosteum into osteogenic and fibrous layers occurred. By the end of the second week, linear fractures were well filled with bone.

At the end of four weeks, activity at the site of injury appeared to have reached an equilibrium. The enchondral and endosteal layer of the capsule forming walls of the perforation showed no osteogenic activity. The connective tissue of the endosteum and that covering the lamina between the coils and in the spiral ligament responded to injury by laying down a meshwork of fibrous tissue, in part of which ossification might begin by the eighth day. Some of the spiral ligament itself was ossified.

In "Observations on the Problem of Otosclerosis," Wittmaack[14] did not concur in the views of some writers that the problem of otosclerosis was concerned only with the labyrinthine capsule. He discussed in some detail arguments previously presented by Nager and Meyer on the similarity of otosclerosis and circumscribed fibrosis of bone, with the conclusion that the otosclerosis disease process might be a limited osteodystrophic fibrosis.

There followed a very profound discussion on the basic characteristics of otosclerosis and osteodystrophic fibrosis, in which the author stated that osteodystrophic fibrosis, while most frequently occurring as a disease process involving the temporal bone, nevertheless occurred far less frequently than the otosclerotic process. Generalized osteodystrophic fibrosis was peculiar to older individuals or was associated with other disease symptoms. Otosclerosis occurred for the most part in young adults, and in individuals who otherwise enjoyed good health.

A critical analysis was made of five cases selected by the author from a group of 53 osteodystrophic fibroses which Nager and Meyer classified as otosclerosis. Profound approval was later given of the research on otosclerosis presented by Gustav Fischer, of Jena, in 1919, and finally a discussion of the vascular behavior in relation to otosclerosis in his own cases. The author maintained it would lead to no solution of the otosclerosis problem to disregard the important basic dif-

ferences in behavior in osteodystrophic and otosclerotic disease processes, or to combine these two very distinct diseases.

"Movements of the Tympanic Membrane and of the Malleus in Normal Cases and in Cases of Otosclerosis" were contrasted by Frenckner.[15] The connection between malleus and incus was not a real joint but a "connection," sometimes osseous, sometimes cartilaginous, and frequently like connective tissue. Cinematographic registration had made it possible to observe and register phenomena of the tympanic membrane in the living being which were valuable in the estimation of certain normal and pathologic conditions. The movements of the tympanic membrane and malleus were filmed during compression and the aspiration of air in the outer auditory canal, and then studied in the enlarged projection of the film. The displacement of the malleus handle could be calculated. The principal movements seemed to be a parallel displacement and, to a lesser extent, a hinge-like movement. In certain cases of otosclerosis, the mobility of the malleus was arrested or practically suspended, which might be due to an otosclerotic change in the malleoincudal joint or in the neighborhood of its surfaces.

"Normal Hearing by Bone Conduction" was presented by Greenbaum, Kerridge and Ross.[16] This paper reviewed the efforts to establish normal hearing standards in 100 patients, 74 males and 26 females, between ages 18 and 25 years, without history of previous ear disease.

The tests were made in a carefully prepared soundproof room, in a carefully observed optimum position over the mastoid. Frequencies of 64 to 8,192 cycles were taken on ascending and descending intensity of sound. The conclusion was that there was a somewhat wide variation even when carefully done on intelligent patients. The variation from the acuity of the inner ear and areas of conduction over the skin-covered mastoid was impossible to estimate. This emphasized the frequent fallacy in rating of bone conduction as abnormal when the determination on the patient is compared with a single normal individual.

Gerlach[17] raised "The Question of the Penetrability of the Cochlea Aqueduct." The majority of writers have agreed that the aqueductus cochlea may be regarded as an open link

between the perilymph cavity of the cochlea and the subarachnoid cavity of the posterior cranial fossa. These conclusions have been reached by chemical and physical research, anatomical and histological studies on humans, as well as introduction of color solutions and injection of Chinese ink into the subdural and subarachnoid cavities in animal experimentation. Cases have been cited of fatal meningitis where infection was transmitted through the aqueductus cochlea. In special case cited, at postmortem a brain tumor involving the entire right half of cerebrum with severe associated edema was found and the aqueductus cochlea was shown to be filled with bone splinters of various sizes, blood elements and blood effusion.

Goodhill[18] reported a "Histopathologic Study of Syphilis of the Ear." Temporal bones of 16 cases of syphilis were studied — eight adult cases of acquired syphilis and eight infantile, prenatal syphilis. These histopathologic changes were quite consistently found:

1. Productive periostitis (luetic) producing invasive fibrosis, followed by ossification of the perilymphatic and endolymphatic channels of the vestibular apparatus. Malformations of the bony capsule were also produced.

2. Bony abnormalities of the stapes, especially of the footplate.

3. Atrophy of the neuroepithelial elements of the cochlear and vestibular systems.

4. Lymphocytic (small round-cell) infiltration of the cochlea and spiral ganglion (miliary gumma).

5. Vascular changes consisting of diapedesis in prenatal cases and obliterative endarteritis in acquired cases.

There was apparently no specific variety of hearing loss associated with luetic aural disease.

Evidence has been presented to show the lack of causal relationships between syphilis and otosclerosis.

Kelley[19] presented a "Study in Presbycusis" — auditory loss with increasing age and its effect on the perception of music and speech. Three problems were investigated:

1. The loss in auditory acuity, which, independent of special pathology, accompanied increasing age.

2. The effect of auditory loss on musical tone perception.

3. The effect of auditory loss on perception of speech.

The first problem concerned persons above 50 years of age. One hundred sixty-eight ears were examined from 64 to 8,192 cycles with a 2A audiometer. The findings were as follows:

1. Up to age of 70 years, hearing was practically normal for frequencies up to 512 cycles.

2. Up to age of 70 years, the loss for 1,024 cycles was small; in the 70 to 79 years period, the loss was only 14 dcb.

3. In age range 70 to 79 years, loss for low frequencies was slight.

In subjects of 50 years of age, presbycusis was already apparent for frequencies of 2,048 cycles and higher. In presbycusis, the loss for high frequencies becomes progressive with increasing age. Most persons above 60 years have lost serviceable hearing for frequencies above 4,096 cycles. This was usually bilateral.

In studying the perception of music in presbycusis, it was found that the elimination of all frequencies above 4,000 cycles did not change the violin tone in a person over 60 years with presbycusis. Such elimination definitely altered same tone in normal ears. The average person past 60 years with presbycusis was not seriously handicapped for vowels, but recognition of consonants was inferior to that of a person with normal hearing.

Koch,[20] in reviewing the "Genesis and Clinical Picture of Acquired Deafness," found that it was extremely difficult to establish deafness as an acquired deafness with any degree of certainty, as in only a limited number can postmortem examination be made. During the past three years he conducted a careful research upon 33 selected cases in which deafness followed hospitalization for other illness.

Normal speech and hearing capacity before the onset of deafness was readily established. The course of disease proc-

esses leading to deafness were carefully studied. Five cases progressed from otitis media to labyrinth involvement with typically significant vestibular disturbance. Middle ear infection was the cause of the remaining 28 cases of deafness. Tables showing the various types of infectious diseases were presented as follows:

I. Meningitis:
- a. Cerebrospinal.
- b. Trauma.
- c. Pneumonia.
- d. Measles.
- e. Scarlet fever.
- f. Grippe.
- g. Typhus.

II. Toxic:
- a. Measles.
- b. Scarlet fever.
- c. Grippe.
- d. Typhus.

III. Myelotic Leukemia.

IV. Congenital Syphilis.

V. Labyrinthitis:
- a. Measles.
- b. Scarlet fever.
- c. Otitis media.

Luscher[21] estimated the "Effects Produced on Hearing Capacities by Means of Tympanic Membrane Weighting." The author described his original research in the following methods of weighting:

1. Burdening of the umbo by increasing weights.

2. Burdening the pars tensa with mercury drops of various weight.

3. Burdening the pars tensa with water drops of various weight.

4. Filling of the auditory meatus with water.

These weights did not affect the organ of Corti nor the nerve channels, hence pure sound conduction disturbances were measured. By this method there were obtained: first, production of pure sound conduction disturbances with deafness for middle and high tones, and diminishing of upper tone limits; second, by means of various functional types of sound conduction disturbances, corresponding air conduction audiograms were registered.

The author gave a detailed description of his method of weighting the membrane, and also detailed description of other methods of weighting with mercury and water drops. The article was well illustrated.

The author believed that auditory loss due to tympanic membrane weighting was far-reaching and dependent upon the precise form of weighting. Burdening of the umbo had a slight influence only on the lower and middle tone ranges. The pars tensa played an important rôle in sound reception and high frequency.

Observations on "Normal and Pathologic Physiology of Sound Conduction Apparatus" were made in a later article by Luscher.[22] The author reviewed the physiologic work of Helmholtz, Bekesy and others, the classic research of Bezold and the more recent works of Guild, of Johns Hopkins, and Hallpike, of Ferens Institute, London.

The author established sound conduction disturbances by mechanical acoustics by the following methods:

1. Increasingly weighting the handle of the malleus: *a.* at the umbo; *b.* at the site of the axis ligament.

2. Increasingly weighting the pars tensa: *a.* by means of quicksilver drops; *b.* by means of water drops.

3. Completely occluding the auditory meatus by Oropax or filling with water.

The author concluded in these tests that it was improbable that typical tympanic membrane weighting affected the nerve apparatus, but by increased liquor pressure caused sound conduction disturbances in the labyrinth and stimulation of the organ of Corti. Bezold has definitely proved increase in labyrinth pressure when the stapes base was dislocated in the direction of the labyrinth.

In weighting the handle of the malleus, he noted that the umbo was the site of greatest, and the rotary axis that of least amplitude. In both cases, loss of hearing occurred in lower and middle parts of the tone series. The upper ends of the series showed only slight effects and extreme upper tone boundaries remained normal. Severe deafness did not follow weighting even to the point of endurance.

In weighting of the pars tensa, either with quicksilver or water, he found preponderant loss of hearing for middle and high tones, with partial diminution of upper tone boundaries. The quicksilver weighting resulted in a more intense effect

than water weighting. Twelve audiograms accompanied the article.

Pohlman[23] presented "A Note on the Greater Disability for Hearing High Tones in Cases of Conduction Deafness." This writer disagreed with the evidence submitted some years ago by Alexander as to a dual conduction of air sounds to the cochlea. This assumed that the low tones were conducted through the ossicular chain, while the high tones passed into the cochlea through the round-window membrane. This writer believed that the drum membrane and ossicular chain was the only effective route for the conduction of air vibrations of all frequencies. The recent work of Crowe and his co-workers citing a type of conduction deafness where there was greater disability for hearing high than low tones has attracted attention. They ascribed the condition to obstruction of the auditory tube with lymphoid tissue, and reported recovery with removal of the obstructive tissue. The author believed that if their theory that a beginning conduction deafness might be found in the lowered sensitivity to high tones and that early treatment might reduce it 50 per cent be substantiated, they have developed one of the most important advances in modern otology.

The author presented cases indicating that this curious limitation in the conduction of high frequencies might be due to causes other than the blocking of the Eustachian tube. He urged the importance of an audiometric examination throughout the frequency range before a definite diagnosis of mixed deafness be made.

"Latent Period of the Crossed Stapedius Reflex in Man" was discussed by Perlman and Case.[24] Reflex contraction of the stapedius muscle to sudden loud sound stimuli protected the cochlear structures from damage by diminishing the amplitude of vibration of the stapes. This reflex contraction occurred in response to nerve impulses arising in the cochlea. During the time necessary for this reflex arc completion, cochlear mechanism remained unprotected. This has been measured for the first time by recording the action potentials of the contracting stapedius muscle. The latency of the human stapedius muscle reflex for a loud tone of 1,000 cycles is about 10:5 seconds. This should account for the increased degree of acoustic trauma through rapidly repeated loud sounds in

the experimental animal, operators of pneumatic hammers, metal stamping machines and rapid-fire arms.

Theissing[25] discussed "Deafness Due to Osteomyelitis." Deafness caused by osteomyelitis was a rare disease, hardly more than 13 known cases recorded. In this accurate estimate, the author included only instances where the labyrinth was injured. Where the middle ear cavity was involved, diagnosis was difficult, but injuries to the middle ear gave a possibly characteristic picture indicating the diagnosis of osteomyelitic deafness. A case was presented showing generalized staphylococcus osteomyelitis, with special locus in the thigh. In connection with this thigh involvement, patient suffered attacks of vertigo with associated tinnitus. Ultimately, the hearing of both ears became very poor. The author stated that osteomyelitic deafness was essentially a disease peculiar to youth. Suggested causes for this condition included embolism, nonsuppurative labyrinthitis and toxic nerve destruction.

Treer[26] presented "New Methods of Testing the Vestibular Apparatus." The caloric procedure in testing the labyrinth has been simplified, but the method remains unchanged, which is the cooling of the semicircular canal by water spray. The author described his experimental study in filling the auditory meatus with cool water. The more marked reaction occurred with water lower than 17° C.

Uffenorde[27] presented "The Significance of X-ray Pictures in Opinions on Hereditary Deafness." In his research, conducted in special schools for the deaf, diagnosis of hereditary deafness could be confirmed in only one-half of the cases studied. In order to lower the level of the undetermined cases, a research was made on the differentiation of type. The Lange-Sonnenkalb and Stenver techniques were used, the former in the study of the pneumatic cell, the latter in showing the designs of labyrinthine structure. X-ray study revealed former middle ear suppuration. The genesis of deafness was noted as hereditary, acquired or undetermined. After exhaustive histologic research, including the embryonic variation, the author advised concentrated efforts on the use of the X-ray in diagnosis of osseous changes going on in acquired deafness. Little is known concerning the pathologic anatomy

of hereditary deafness. The author cited the arrested development of sound perception apparatus, while in hereditary deafness he called attention to the malformation of the bony modiolus, faulty differentiation of Corti's organ, peripheral auditory nerves and ganglion cells. X-ray evidences of pneumatic disturbances could not be considered proof of acquired deafness, but were a contributing factor in the research and assisted in the determination of type of deafness. Paper was clarified by six illustrations.

Williams[28] made a Preliminary Report on the Loss of Hearing from "Otitis Media and Orbital Cellulitis Complicating Scarlet Fever." He reported on four groups:

1. With no known involvement of the ear: Fifty-six subjects had no known involvement, with an average loss of hearing of 7.9 per cent. Three patients had moderate loss for high tones.

2. Patients with earache but no discharge: Fifteen patients belonged in this class, with an average loss of 5 per cent in hearing.

3. Patients with discharging ears: Twenty-four patients had discharging ears during the scarlet fever. Average loss of hearing was 10.5 per cent, and four had moderate loss for the higher tones.

4. Patients requiring mastoidectomy: Nine patients required a mastoidectomy operation. The average loss of hearing was 9.9 per cent.

Further comments:

1. Loss of hearing was greatest in subjects with previous discharging ears.

2. Removal of the tonsils had little effect on the loss of hearing.

3. History of sinusitis had little effect except when associated with otitis media.

Zeckel and Van Der Kolk[29] reported on the "Comparative Intelligence Research on Groups of Children Afflicted with Hereditary Deafness and Those with Normal Hearing Capacities, by Means of Porteous Intelligence Tests." Difficulties of

sound perception in the congenitally deaf were apt to be accompanied by difficulties in perception. This condition led to psychic changes in later years, even up to general psychoses with paranoic manifestations.

The authors in this research limited themselves to congenital deafness, and selected the so-called performance tests. It was their opinion that tests which required accomplishment of simple tasks were far more effective than those which depended upon a considerable vocabulary. They studied 100 congenitally deaf children and 100 with normal hearing capacity, with an age range of 7 to 14 years. It was found that the temperament played a considerable rôle. This was admittedly a small group for deduction. In the early years (age 7 to 11), the deaf children showed a considerably retarded intelligence index in comparison with the normal group, but in the later years (age 11 to 15), this index rose appreciably. While the results of the research were not conclusive, everything indicated that lack of auditory perception influenced the intelligence development. More detailed research should be undertaken to determine to what extent the deaf child was conscious of his defect, how much this influenced other functions, whether it tended to create a world of phantasy or to stimulate memory, and whether there was compensation by increased efficiency of the other senses.

Van Dishoeck[30] presented the "Pneumophone and Tubal Resistance Measurements Among Aviators." In this article, experimental pressures in the Eustachian tube were technically discussed. This principally concerned resultant effects of pressure on aviators and caisson workers, and is simplified in the following article by Zollner.

Zollner[31] discussed "Obscure Disturbances of Pressure Balance in the Middle Ear." The author had discovered earlier by means of tubal resistance measurements the disturbance of pressure balance in the middle ear recorded by Van Dishoeck through the use of the pneumophone. Both authors attained the same results, though approaching the problem by different angles. The applications of these different pressure tests on the external canal, tympanic area and nasopharynx presented chief interest in their relation to aviation and caisson workers. One interesting observation was that the normal ear endured slight variations in pressure for some

time without evidence of disturbance. It might be concluded that up to a certain point the inner ear muscles were able to compensate for a burdened chain of auditory ossicles. When, however, the burden became too great and symptoms of inflammation arose, this assimilation was no longer possible, and disturbances occurred in the form of tinnitus aurium or deafness.

Bertoin[32] discussed "Influence of Cranial Trauma on the Evolution of Otospongiosis." Otospongiosis (otosclerosis) did not have an invariable clinical picture. It was a dystrophy of endocrine origin, as shown by the almost invariable increase in basal metabolism (Rebattu and Mounier-Kuhn) with disturbance of the sympathetic system as cause or result. Otto Mayer, of Vienna, stated that otosclerosis began by bony resorption. The site was clearly demarcated and the histologic structure of the tissue resembled osseous scarring. This was often accompanied by blue sclerosis and evidence of bone fragility. Pressure prevented normal movement and elasticity of the two layers surrounding the labyrinth, and small fissures were formed. Besides the mechanical, there were constitutional factors. Portmann agreed with Mayer that trauma was one of the numerous causes favoring the development of otosclerosis. Pregnancy, lactation, chills, over-fatigue and residence near the sea were among the factors of accelerating its development, and it was simply a question of whether or not cranial trauma should be added to the group. The author presented two cases histories.

General conclusions could not be drawn from two cases, however. It was noted that violent cranial trauma was immediately followed by marked aggravation of auditory deficiency. This was followed by slow improvement, spontaneous or influenced by medical care and treatment, and finally hearing became stabilized at or below the previous level. For medicolegal purposes it would always be well to wait for several months before evaluating the extent of injury. It would be extremely difficult to determine the pathogenesis of such phenomena in the absence of pathological examination.

A "Contribution of Sound Direction Determination in Case of Intact Cochlea and Defective Vestibule, Conjointly with Clinical Investigation of Tumors of the Fourth Ventricle" was presented by Guttich.[33] The author stressed the observa-

tion that associated otologic symptoms of a normal cochlea and vestibular deviation always suggested the possibility of existing disturbance of the ventricular system. A typical case of such occurrence was presented. In this particular case, examination showed the third ventricle to be completely filled with tumor substance which had been developed in some degree in the fourth ventricle. The peculiar symptomatology in this case was presented by the author and, incidentally, the phenomena of disturbance in sound direction determination was explained.

Arnold,[34] in a "Contribution to Knowledge of Localization of Cochlear Disease," called attention to certain cochlear changes in the C^5 tone range as related to so-called isolated vestibular deviations. These were single or bilateral deviations in the vestibular equilibrium apparatus, with associated deafness or impairment of hearing.

Uffenorde, Langenbeck and Tanturri had noted similar fifth octave circumscribed changes. Experimental research on sound disturbances indicated that any irritation or fatigue symptoms could normally cause tinnitus aurium in the fifth sounded octave. The author suggested, too, the connection between the C^5 gaps and defective enunciation of sibilants.

Under "Recent Progress in the Treatment of Deafness of Otosclerotic Origin," the work of various authors was reviewed by Beck and Guttman.[35] Gray and Goldstein reported improvement in hearing and diminution in tinnitus by intratympanic injections of thyroxine. Most other observers, however, have given up this form of therapy because of lack of consistently good results. Reinisch reported that otosclerotics had an increase in deafness under epinephrine, and improvement under pilocarpine. This improvement lasted 10 to 12 days and could be repeated by further injections. Mortimer, Wright, Collip and Thomson reported improvement in otosclerosis by daily insufflations of 1 cc. of oil containing 1,000 international units of estrin. Sourdille, Holmgren and Lempert have devised surgical techniques for otosclerosis by making fenestra in the semicircular canal. Lempert maintained patent fenestra in 22 out of 23 cases, with improvement in hearing in 19 and failure in four cases. Hayden described modern hearing aids which had contributed greatly in the otosclerotic problem.

Albrecht[36] presented "Histologic Findings in a Recent Case of Recessive Deafness, and a Study of Its Limits as Compared to Acquired Deafness." He reported a patient under observation for five years with complete deafness on one side and diminished hearing on the other. This patient died, as was reported, from tuberculosis; complete postmortem studies were made of all structures in the internal ear and its connections. The findings on both sides, one of hereditary and the other acquired deafness, indicated similar changes in the organ of Corti. Definite evidences of residue of old inflammation in case of acquired deafness were stressed. Marked changes in the cochlear muscles and in the nerve trunk were regarded as clear signs of hereditary deafness. For valuable deductions, complete family history must be in hand and the research must include the brain, nerve trunk and peripheral organs.

That malformations and traumatic injuries could not be proved definite causes of deafness was the opinion brought out in the discussion.

Engstrom[37] wrote "On the Frequency of Otosclerosis." He reported on sectional material of 61 temporal bones from 34 unselected individuals, between the ages of 30 and 90 years:

1. Otosclerosis was found in four cases.

2. In these four cases, otosclerosis was bilateral.

3. In two of these cases the otosclerosis was localized to the region of the oval window, with exostosis processes and thickening of the annular ligament. There was no bony ankylosis.

4. In the other two cases, otosclerosis was diffuse in the labyrinthine capsule, without link to structures important to sound conduction or perception. There had been in these cases no reported history of cochlear or vestibular symptomatology.

Harkness[38] reviewed the "Problem of Deafness." In 1932 there were 3,000,000 deafened school children in the United States. There were three types of deafness — conductive, nerve and otosclerotic. Fundamental tones of speech range were from 256 to 2,000 cycles. The decibel, the unit of meas-

ure, represented the smallest change in sound the human ear could detect — the sensation unit.

A proper understanding of hearing required entrance into the field of electrodynamics. The improper aeration of the upper respiratory tract was the chief cause of an abnormal conductive mechanism.

Important in causing acquired deafness, and in this order, were meningitis, measles, scarlet fever and influenza; syphilis had not proved an important factor. Severe deafness after measles and scarlet fever had usually proven nerve deafness.

The psychology of the deaf was an important matter; hence the importance of hearing aids and lip-reading.

"Hearing Acuity and Stammering" was analyzed by Harms and Malone.[39] Speech defects in pupils of elementary grades have been estimated at from 4 per cent to 18 per cent, which meant that 1,000,000 children had defective speech. Speech involved a mental motor mechanism and a proper receiving mechanism; therefore, defective hearing was a legitimate cause for defective speech.

The authors emphasized the importance of testing the hearing acuity of all school children before the end of the first grade. This was fully as important as the visual acuity tests. At this age, existing pathology could be cared for. Stammering was found to be rare in cases of total loss of hearing, and more frequent in those with approximately 50 per cent loss. The relation between stammering and partial hearing loss thus became most suggestive.

Littler[40] discussed "Resonance in the External Auditory Meatus." A knowledge of the extent of resonance in the auditory meatus gave information of value as to the magnitude of effective amplification hearing aids for the deaf. The author suggested replacing the air in the meatus by some other gas in which the velocity of sound differed from that in air. His experiment showed that there was quite an appreciable pressure magnification (about three times) due to meatus resonance. The flatness of resonance indicated that energy was absorbed by the material and mechanism of the ear. Undoubtedly some of the energy was consumed in overcoming viscous forces in the middle and internal ear, and

some of it may have been transformed electrically in the inner ear.

Ohma[41] wrote concerning the "Changed Relationships of Disease in the Endolymph Cavities and Canals." Referring to the experiments of Wittmaack, Hyrtl, Kibala, Miyamoto and Uyama, he stated:

1. The perilymph came from the cerebrospinal fluid going mainly through the aqueductus cochlea to the labyrinth.

2. Collapse of the membrane of Reissner might not only arise in the labyrinth but could be brought about by closing off the aqueductus cochlea.

3. Closing of the aqueductus cochlea could be considered a cause of deafness at birth.

This article discussed the various effects of pressure, venous transmission and anatomic variations in the labyrinthine chambers, as well as regulating mechanisms and valves. The author stated that dilatation of the cavities of endolymph could not only be caused by labyrinthitis, but also by disturbed penetrability of the endolymphatic duct system, and still further by disturbance between endolymph and perilymph pressure. The utricle and semicircular canals could keep their normal pressure independent of pressure in saccule and ductus cochlea.

Ruttin[42] gave an "Explanation of Syphilitic Symptoms During an Attack of Acute Otitis." The author presented a case in which syphilitic symptoms suddenly appeared during the course of an acute otitis and as quickly disappeared with the cure of the otitis. While in this case of deafness following an attack of scarlet fever, sensations of vertigo appeared when placing the finger in the left ear and quickly removing it, and Wassermann test for suspected congenital syphilis and eye examinations were both negative, later symptoms demonstrated syphilis. In regard to this case, one author accounted for the syphilitic symptoms as the loosening of the annular ligament by infiltration; another, that they were caused by a gumma in the region of the stapes. The author accepted the annular ligament theory as the more plausible.

Riecke[43] presented "Hereditary Deafness and Its Significance in Daily Practice." The author considered deafness not

a disease *per se* but a resultant condition due to embryonic defects of external factors. He classified the following types of hereditary defective hearing:

1. Recessive sporadic deafness.

2. Progressive inner ear deafness.

3. Endemic deafness as in cretinism.

4. Deafness due to syphilis.

5. Deafness developed at birth from trauma or forceps injury.

For the protection of posterity the author counseled most careful histological investigation of temporal bone, brain, central nervous system and peripheral nerves with recorded clinical findings and family history. Where there was an established history of hereditary deafness, propagation should be avoided.

Baer[44] presented a paper on "Otitis Media and Dietetics." Inflammatory changes in the middle ear were chiefly due to infection. Chemical and physical factors usually affected the inner ear. The virulence of the bacterial element varied greatly in different locations and conditions. The author believed that various diseases, especially those involving metabolic changes, favored the outbreak of infection, and that a poor evaluation of dietetics predisposed the body to infection and subsequent middle ear inflammation.

The author discussed the various vitamin deficiencies and the value of the different types, especially in otitis media.

Buchband[45] discussed the "Symptomatology of Mastoiditis Following Bathing-Otitis." Cases of water infiltration during bathing which resulted in infection in the middle ear fell into two groups:

1. Diving cases, when water entered the mouth and nose, reaching the middle ear through the Eustachian tube, the tubal-tympanic infection group.

2. Traumatic cases (blow on the head, as by a ball in water sports), with a rupture of the tympanic membrane and water reaching the middle ear through the external auditory meatus — the tympanic infection route.

Twenty case histories were described in detail. The author concluded that mastoiditis following bathing-otitis showed a more serious course than mastoiditis of other etiology, especially where infection occurred through the external auditory meatus and laceration of the tympanic membrane.

Goebel[46] reviewed "Electrokinesis and Hearing Perceptions." The author, after elaborating upon the technique of stimulating sound perceptions through alternating currents, discussed the question as to whether the auditory stimulation was due to irritation of the labyrinth or the auditory nerve, but concluded that the auditory irritating effect resulted only from direct stimulation of the auditory fibres. He presented another opinion that perhaps the hearing sensations which arose from the alternating current stimulation might be due to mechanical displacement of the organs of the labyrinth.

Jannulis[47] reported "Two Distinct Types of Otitis." In this, the author presented his observations on the following types: *1.* Serous otitis externa; and *2.* acute otitis media affecting auditory nerve. In the first group studied, between the ages of 25 and 50 years, the author discussed infection of the external canal and chemotherapy. In the second group, the basis was considered a toxic infection of the cochlea, through a micro-organism from the middle ear.

Richter[48] presented a "Critique on Deafness." The author regarded deafness as a symptom, not a disease *per se*. Frequently a patient consulting a physician for illness other than that occasioned by deafness would complain of auditory disturbances. An exhaustive study of hearing capacity should be made in order to avoid the danger of having serious complications charged to diagnostic errors. External, middle and internal ear conditions should be analyzed. The author impressed the following on his students: He who wished to treat ear disease must be able to readily evaluate all the peculiarities of the tympanic membrane. He stated that deafness was always conditioned by disease of the labyrinth or of the auditory nerves. Most inflammation of the labyrinth originated from the middle ear. Tuning fork tests and careful family history were of utmost importance. The auditory disturbance might be a symptom of some menacing disease, and require the greatest diagnostic care.

Schwarz[49] commented on "Otosclerosis and Body Structures." The author maintained that diseased surrounding tissue restricted itself to the bones of the labyrinth capsule, that the appearance of otosclerosis was associated with fragility of bone; the rôle of embryonic tissue development in its various phases was discussed. The statement was made that in 80 per cent of typical cases of otosclerosis, the tympanic membrane was extremely tender. Manifestations of body weakness was emphasized. Apparently this was given as a preliminary investigation.

"Deleterious Effects of Noise in Industrial Pursuits" was presented by Weyrauch.[50] It has been well established that continued noise caused lowered hearing and led to complete deafness. Both autopsy findings and animal research have proven that intense noise could completely destroy the organ of Corti in the cochlear duct. In the case of typists who used dictaphones, marked depreciation of normal hearing occurred in one to three years. A large percentage of accidents have been caused by noise; first, because the worker was deafened by the noise; second, because the warning signs were not heard when submerged in prevailing noise. The practical value of protective measures to prevent deafness among railroad and industrial workers has not yet been proven.

The question, "How Do Teacher and Physician Co-operate in Work for Deaf-Mutes" was analyzed by Baldrian.[51] The author reviewed the great handicap of the deaf-mutes in acquiring lip-reading and the mastering of speech. In addition to skilled instruction in this, the aid of a physician was also necessary. He recalled the Nuremberg Hygiene Congress of 1904, when proposals were made for the establishment of institutions for deaf-mutes. To be successful, these required the co-operation of general practitioner, otologist, oculist and dental surgeons.

"The Effect of Lesions in the Medial Geniculate Bodies Upon Hearing in a Cat" was reported by Ades, Mettler and Cullen.[52] It had been reported that the various segments of the cochlea respond differently to acoustic stimuli, the higher frequencies being resonant in the basal coils, and the lower near the apex. Two of these authors have made a preliminary report tentatively confirming these electrical results

with hearing tests by the use of the conditioned response technique in cases of localized lesions in the cochlea.

Functional evidence of localization of tone at any level above the cochlea was conspicuously lacking. Meagre clinical records indicated the possibility of partial tonal deafness in consequence of localized injury to the temporal cortex. The medial geniculates, being the final way station from cochlea to cortex, provided an ideal site for determining by what pathways cochlear impulses were transmitted to the brain.

Results of these studies:

1. Small localized lesions in the medial geniculate bodies produced markedly unequal losses in auditory acuity at the several test frequencies.

2. Cats with lesions in corresponding areas displayed the same distribution of hearing losses. In this way loci for the several frequencies have been established as follows: 8,000 cyles, dorsal region; 4,000 cycles, anterior region; 2,000 cycles, lateral region; 1,000 cycles, posterior region; 500 cycles, medial region. It thus appeared that the several loci followed a linear course beginning with high frequencies in the dorsal section and circling downward to the neutral region, where the lower frequencies appeared.

3. Functional evidence indicated that the medial geniculate body carried a projection of the organ of Corti, each part of the latter being connected by bundles of fibres with corresponding areas of the former.

4. Since different frequencies traversed the geniculates by separate pathways, it followed that pitch discrimination occurred at lower levels (cochlea).

GROUP 2. MEDICAL THERAPEUSIS AND SYSTEMIC READJUSTMENT.

"A Thyratron Inflector, Its Behavior with Certain Vowels and Its Use in Instructing Deaf Children" was presented and carefully explained by Sterne and Zimmerman.[53] In the introduction they explained that the important aim in teaching deaf children was to make their speech intelligible, well pitched and pleasing to the ear. The deaf person had to rely mainly on kinesthetic and touch sensations for perception of

pitch. The underlying principle in educating the deaf was to substitute the use of other senses (touch and vision) for perception of such stimuli as were normally perceived by the ear.

It has been important to provide deaf pupils with some means of enabling them to see positive and definite effects produced by their voice sounds. The apparatus described had a vertical row of lights to flash in accordance with pitch variation of the voice. With the higher pitch the greater number of lights respond and voice and its inflection are translated into visual symbols.

In using the instrument, voice is directed into a crystal microphone and the sound waves are transformed into electrical impulses which activate the thyratron tubes, which in turn cause seven neon lamps of this vertical series to light. A low frequency sound will cause the lamp at the bottom of the column to flash, and a sound of higher frequency will cause successively higher lamps to flash.

The machine was intended for all types of voices; therefore, three different values of tone frequency can be regulated for low, middle and high voices.

So that children may feel the vibrations of their speech sounds while they are watching the lights, a permanent magnet speaker is included.

One of the most difficult tasks faced by the teacher of the deaf is getting a change of pitch in the voice of the deaf pupil. Change of pitch is especially essential to the deaf child since it is the first step in teaching the correct inflection of speech. Without a change of pitch, the voice of the deaf child is wooden, lifeless and monotonous.

Previously, it has often taken months to accomplish this change of pitch. With the thyratron inflector this time has been greatly shortened. In several instances the pupil has given a change of pitch after one or two practice periods at the machine.

From a historical standpoint, Alexander Graham Bell was apparently the first to realize the possibility of instruments translating sound into light. In 1873, he became interested in the "manometric capsule" of Koenig, which in effect presented a flame moving up and down with rapidity, propor-

tional to the voice vibration. Scripture reported on the use of the "manometric" capsule in correcting the speech of the deaf. Allusion was made to Scripture's strobilion, devised in 1912, which also depended upon a flame indication of the frequency of tone; to the audiophone stroboscope described by Ferreri, and discussed the Coyne instrument in which electric lamps were used to indicate pitch level.

An important section of the paper was devoted to the characteristics of speech sounds. This was highly technical and should be consulted in the original rather than in a condensed abstract, which could hardly be adequate.

Under the heading, "The Inflector in Use," additional observations were made upon the vowel sounds and a careful description was given of the actual use of the inflector in the practical instruction of the deaf.

In review of results and conclusions, the authors believed that while considerable time must elapse before the value of the inflector could be accurately estimated, it had already appeared that the instrument was extremely helpful, and it was found that children without exception were interested and eager in helping themselves. It was found that very young children, even of 6 and 7 years of age, could derive considerable benefit.

In closing, sincere expressions of appreciation were made to Dr. Max A. Goldstein, of the Central Institute for the Deaf, and Prof. Glasgow, of Washington University, for their help and constant encouragement in the development of this work. The former presented a careful paper on this subject under the title, "Defective Speech in Relation to Defective Hearing," before the American Laryngological Association in 1939, and published in their *Transactions* of 1939. A reference to this, although not strictly a paper on progressive deafness, would seem appropriate as it further clarifies the above abstract.

McCaskey[54] commented on "Speech and Hearing Defects." He first quoted Newhart in the statement that among 50,000,000 children of school age in the United States. 60,000 were totally deaf, 3,000,000 hard-of-hearing, and 4,000,000 defective in speech. The author stated that speech defects occurred eight times as frequently among deafened as normal people. Defects of speech and hearing were closely related and it was

estimated that one-tenth of the people in the United States were speech cripples of some time.

The author urged the combined efforts of family physician, otolaryngologist, psychologist and trainers of speech and lip-reading. The audiometer, recognition and treatment of nose and throat infections, and mechanical aids were all correlated in the help to these patients.

Mitchell[55] discussed "The Education of Deaf Children." The Education Act of 1921 in England required local education authorities to provide educational facilities for the deaf. Compulsory education for this group covered ages of 7 to 16 years. The Act of 1937 lowered the beginning age to 5 years. Several grade divisions were made from children able to benefit in the ordinary school to the totally deaf. In testing gramophone audiometers (spoken word) and in more severe impairments, pure tone audiometers were used. The multitone, a sound amplifier which filtered out certain frequencies and intensified others, was used for teaching six to eight children at a time.

Baer[56] wrote on "The Effect Vitamins Had on Otosclerosis." To Politzer belonged the distinction of being the first to recognize the anatomical peculiarities in this disease. Stapes ankylosis was recognized early, but these characteristic bony processes could occur in other parts of the bony capsule and be quite independent of the presence of stapes ankylosis. There was divergence of opinion as to the course of otosclerotic processes but it seemed probable that in otosclerosis there was formation of new bone rather than transformation of old labyrinthine bone. The knowledge of the genesis of otosclerosis was very limited; in part, at least, it was an hereditary disease, but indications pointed also to constitutional factors. Both the spasmophilic anomaly theory of Kobrak and hormone significance must also be considered.

The proof of connection between otosclerosis and metabolism disturbance was still undetermined. In therapy, the mechanical treatment, such as massage, could be carried out in only a limited degree. Reports of improvement of stapes ankylosis by operative measures have been recently made. Max Meyer reported improved hearing following suboccipital puncture, and Holmgren and Sourdille have claimed improved

hearing by decompression of the peri- and endolymph. In medical therapy, phosphorus, iodine compounds, etc., apparently had value but this was not yet fully established. Russian research on lysates and vaccine therapy reported from Uruguay was under investigation.

Vitamins, single or in combination, were under research study as to their effect upon bone processes. This author's work has been principally devoted to the influence of vitamins B and C on otosclerosis. Though the results have not been of outstanding value, he found that medical preparations were enhanced when given in combination with vitamins, and he interpreted a significant connection between otosclerosis and vitamin C-deficiency. He obtained good results only in cases of tinnitus aurium and never in simple otosclerotic conditions. He attributed his comparatively negative results to a sharper diagnostic differentiation of otosclerotic conditions. He believed that ultimately vitamin treatment could be proved a great aid in coping with otosclerosis.

Selfridge[37] made a preliminary report upon "Nicotinic Acid and the VIIIth nerve." Nicotinic acid, one of the factors of vitamin B_2 complex, has recently been brought forth as a cure for pellagra. The filtrate factors of vitamin B complex were studied by the author in several cases of chronic progressive deafness because the bone conduction curve showed a marked drop at 1,024 and 2,048 cycles. Its use was suggested by Covell's discovery of the rapid degeneration of the VIIIth nerve in vitamin B deficiency in dermatitis chicks. The author claimed that the use of this material which contained some B_6 and nicotinic acid, apparently straightened up some portions of the conduction curve dips. The use of nicotinic acid in deaf humans after the age of 55 years would seem to be justified, rather than B_1.

The author's observation showed a more rapid improvement in air conduction and bone conduction audiometric curves with nicotinic acid rather than with B_1 (thiamin) or B_2 (riboflavin). He also observed that permanent control in hearing could be obtained only in permanent changes in dietary habits, even though vitamins were added. It is quite important in all cases of beginning high tone deafness to consider possible allergy, drugs, syphilis, arteriosclerosis,

water balance, hypothyroidism, and have a thorough physical examination.

The most striking reaction following injection of nicotinic acid, particularly in vasomotor unstables, was marked flushing of the skin, accompanied by a sensation of heat, especially in the face.

The author has accumulated some evidence that this vasodilator effect not only influenced the VIIIth nerve but appeared to play a part in removing calcium in the adhesions around the footplate of the stapes, in chronic progressive deafness and in early cases of bony fixation in true otosclerosis.

Selfridge[58] presented a "Comparative Study of the VIIIth Nerve in Relation to Thiamin Chloride and Nicotinic Acid." The dual nature of certain vitamin B complex deficiencies was becoming more evident. This has been observed particularly in pellagra. This present study was based on the urinary analyses made available after Sebrell's statement on the output of B_1 and porphyrin.

In a recent study of 30 cases to whom nicotinic acid, nicotinamide or sodium nicotinate were used in treatment, the improvement in many cases of VIIIth nerve deafness were striking.

In a series of cases of conduction deafness in young girls and women, age from 15 to 40 years, who showed menstrual irregularities and to whom estrogenic substances had been given with definite improvement not only in the hearing curve but also in relief of tinnitus of the roaring type, nervousness, menstrual irregularities, etc., the B_1 output was low and porphyrin was increased in 50 per cent of the cases. In many instances the addition of B_1 and nicotinic acid further improved the hearing curve.

The VIIIth nerve was a peripheral nerve and the cochlea was its end-organ and was subject to the same degenerative changes as were found in various disease and toxic conditions related to neuritis. Underlying nutritional deficiency might explain these nerve changes. The dose of thiamin chloride was now controlled by urinary tests instead of by estimation.

Any obvious dysfunction of the endocrines should be cared for, and data in this paper suggested strongly that nutritional

deficiencies offer the best solution for handling the degenerative process involving the auditory nerve.

Selfridge[59] presented a third paper in his series, entitled, "VIIIth Nerve High Tone Deafness from a Nutritional Standpoint." The author first discussed the thesis presented some months ago that in the early stages of obstruction of Eustachian tubes of children, the hearing was more impaired for high than for low tones, and its possible etiology. The author noted that in 40 cases of conductive deafness to whom estrogenic substances were given, all showed an audiogram drop in the high tone portion of the scale.

Most otologists have looked upon nerve changes as secondary to infection, yet several authors had agreed that "vitamin deficiency should now be considered a factor of possible importance in the mechanism producing many cases of degeneration of peripheral nerves."

The author discussed the efficacy of vitamin B complex and the pickup in relapsed hearing by thiamin and nicotinic acid.

The low B_1 urinary output and increased porphyrin was apparently an index to the use of nicotinic acid.

The interrelationship between vitamin B and the endocrines, especially the pituitary, was discussed in relation to water balance, and the adrenal cortex as the stabilizer of electrolytes, especially sodium, potassium and magnesium. This interrelationship might offer an explanation of Ménière's disease.

The writer discussed in detail the technique required for determining thiamin chloride and porphyrin, and stated this report was presented to point the way for study of other factors involved in the degeneration of the auditory apparatus.

Babbitt[60] presented a "Study of Medical and Surgical Aids to Hearing." Four fundamental topics were presented for consideration:

1. Medical management in deafness.

2. Readjustment in upper respiratory tract from physiological and focal standpoints.

3. Deafness and chronic otological infection.

4. Otosclerosis and fistulization surgery.

The author reviewed the important recent contributions which aid analysis and treatment of deafness. The examination of a patient with a view to medical readjustment must be more painstaking and with broader scope than the routine upper respiratory review. It must include accurate tests for degree and type of deafness, with the last word on familial and environmental information. From this basic study every possible information as to nerve damage, neural influence of previous acute infections, drug poisoning, nutritional, vitamin and dietary imbalances, should have been obtained. Due appreciation of the convincing influence of allergy as portrayed by Dean, the prenatal drug influence analyzed by Covell, Dorothy Wolff, Taylor and Mosher, must be fitted into the picture, and allowance made for the psychological factors presented by Friesner.

In practical therapeutic suggestion, the author emphasized on the medical side a complete nutritional readjustment and reported the results of routine administration of vitamin B complex in a group of 100 office patients.

In the readjustment of the upper respiratory tract, the author supplemented the customary emphasis on the removal of all focal infections in sinuses, teeth, the Waldeyer tract, pyriform fossa, middle and external ear, and registered a plea against the traditional condemnation of tonsil, sinus and septal surgery as a possible aid to hearing.

In the deafness incident to chronic auricular canal and middle ear infection, endaural surgery, particularly in recurrent cases, offered optimistic possibilities.

Fistulization surgery for otosclerosis in the work of Holmgren in Sweden, Sourdille in France, and Lempert with his pupils in New York, was carefully reviewed, as well as the round-window block of Hughson. The actual details of these methods have been more than carefully reviewed elsewhere during the past year.

"The Treatment of Ménière's Disease with Histamine Administered Intravenously" was presented by Shelden and Horton.[91] Ménière's syndrome has been described as the recurrent attacks of sudden, severe vertigo, with nausea, vomiting,

tinnitus and deafness of unknown etiology. The authors believed that the site of the lesion was probably in the inner ear itself. The factor most likely responsible for Ménière's syndrome was local alteration in the permeability of the capillary wall with secondary local edema. This edema might present a hemorrhage of blood serum alone. Histamine was an important agent for defective capillary permeability. The authors believed that vertigo was the essential feature in this syndrome, and the presence of tinnitus and deafness indicated that the process also involved the cochlea.

This article made a preliminary report of the treatment of 15 Ménière patients with histamine. Four of these 15 received histamine subcutaneously, and 11 intravenously. In the intravenous injection, 1.9 mg. of histamine acid phosphate dissolved in 250 cc. of physiologic salt solution was given. It took one and a half hours to administer the drug. In some cases the same amount of histamine was given on two or three successive days.

The first patient so treated after three weeks' duration of severe Ménière's disease, and confinement to bed, promptly recovered and remained well. One patient had been vomiting for 15 hours before the histamine was started. All cases responded in a spectacular manner. Disappearance of tinnitus and loss of hearing was also reported in these cases.

"Potentialities of Vitamin C Treatment in Cases of Labyrinth Deafness and Tinnitus Aurium" were reviewed by Szolnoky.[92] He presented 50 case histories as proof of favorable results in the use of standardized doses of vitamin C in labyrinthine deafness and tinnitus aurium. These were grouped as follows: *1.* Otosclerosis, five cases; *2.* chronic middle ear catarrh, five cases; *3.* labyrinthine deafness, 40 cases.

In the otosclerosis group the author claimed that irritating symptoms of tinnitus aurium were improved under local treatment and injections. No improvement in hearing was expected.

In the chronic middle ear group, there was relief from annoying tinnitus aurium and some improvement in hearing as long as treatment was continued. The remaining 40 cases of labyrinthine deafness were predominantly arteriosclerotic, with some neurotics and some industrial cases. The author

claimed some remarkable instances of improved hearing. Careful diet and correct living were counseled as important adjuncts.

"Therapy of Tinnitus Aurium. The Effects of Hypertonic Magnesium Sulphate Solution" was elaborated by Ioannovich.[63] The author first discussed local and general causes. The local causes were: dystrophic, inflammatory, neoplastic, degenerative. General causes were: vasomotor, organic vascular, contagious or toxic. Tinnitus aurium caused by vasomotor and vascular disturbance was often accompanied by severe head pains due to increased perilymph pressure. Lumbar puncture often completely cured this. In chronic functional disturbances, marked improvement often resulted from intravenous injections capable of producing strong vasomotor reaction. In the laboratory at Athens, they demonstrated this by the use of an intravenous injection of 50 per cent solution of magnesium sulphate. The author outlined the result in 50 otologic cases, 19 males and 31 females. His classification included: 19 cases of vasomotor disturbance of the labyrinth; one case of arteriosclerosis of the labyrinth; nine cases of middle ear disease with disturbances of conduction; six cases of otosclerosis; 15 cases of neuritis of the auditory nerve.

Eliminating the nine cases of conductive disturbance, the author claimed that the 41 remaining cases of labyrinthine affection gave the following results: cured, 13, or 31.7 per cent; improved, 20, or 48.78 per cent; unchanged, eight, or 19.51 per cent.

The author concluded that buzzing in the ears due to vasomotor or vascular disturbances of the labyrinth was cured in 50 per cent of the cases by injections of magnesium sulphate solution. Buzzing in the ear due to otitis, syphilis and otosclerosis could only be helped in exceptional cases.

Erodi[64] presented "Facts About Vitamin Therapy in the Therapeutics of the Throat, Nose and Ear." Vitamins were in use by the Egyptians in 1500 B.C. Vitamin A increased resistance of the epidermis in the prevention of infection. It was valuable, therefore, in the mucous membrane of the nose, accessory sinuses, tympanic cavity and teeth. Its use in the form of a salve also had value.

Vitamin B was especially indicated in diseases of neuralgia, neuritis, and disturbances of innervation from toxic or rheumatic origin. It was appropriate for facial and postdiphtheritic paralysis. Vitamin C, while important for nose, ear and throat therapeutics, by increasing resistance, had postoperative value and hemostatic effect in bleeding. Vitamin C was indicated in ulcerative processes, especially those of unknown origin and Vincent's angina.

Vitamin D was an important factor in phosphorus and calcium metabolism. Vitamins E, F and H were not yet used in rhinolaryngology.

Seiferth[65] wrote "concerning the Treatment of Otosclerosis." The author described otosclerosis as a constitutional, hereditary disease, characterized by progressive, degenerative processes which result in inner ear deafness. This diagnosis was based on hearing tests and genealogic tables.

The author contended that mechanical inflation of the Eustachian tube and pneumatic massage of the tympanic membrane were only favorable if there was associated tubal affection. In otosclerosis with involvement of the stapes base. mechanical treatment was futile. The author discussed favorable results in the effect of phosphorus medication and lime injection intramuscularly or intravenously. He has also conducted experiments on mineral metabolism in relation to its reaction on the endocrine system.

After reviewing the therapeutic efficacy in hormone disturbance, he discussed briefly surgical measures with their possible result of complete deafness, and emphasized the importance of psychotherapeutic direction of these easily depressed patients. He made the comment that in advanced progressive deafness, lip-reading, far too little appreciated, markedly compensated for loss of hearing.

Ebermeier[66] discussed the question, "Is Labyrinth Concussion a Frequent Result of Skull Fracture, and How Can It Be Influenced Medicinally?" The author considered labyrinthine concussion as closely linked with cranial injury, as labyrinthine symptoms occurred in such a large percentage of skull fractures. In his conclusion, the author believed that differentiation of labyrinthine symptoms and common cerebral symptoms was difficult in the acute stage of injury, but

was quite possible in the later stages. He further emphasized the efficacy of the Monotrean preparation, quinine for hyperemic and papaverin for tonic effect, in the treatment of vertigo arising in the case of labyrinthine concussion.

Koch[67] presented "Views and Treatment of Senile Deafness." Since hormone treatment controlled many difficulties with which senile deafness was bound and was responsible for improvement in numerous cases, the author concluded that they could accomplish very real service in this type of deafness. He referred to the research of von Fieandt and Saxen, who attributed senile deafness to two factors:

1. Senile atrophy of the cochlear ganglia.

2. Angiosclerotic degeneration of the inner ear.

He advocated and enumerated hormone preparations which were believed to be of value. The list included progynon, proviron, memformon, unden, testoviron and hombreal. Injections were given intramuscularly, starting with large doses and gradually decreasing them. Tables were presented showing the results of the hormone treatment.

As the author stated, it was assumed from the results of this research that generated gland hormones played a very real rôle in senile deafness, although vitamin B was a very important factor.

"Penetration of the Inner Ear by Mercurochrome" was reported by Ross and Hamilton.[68] In 1908, Van Rossen was the first to anesthetize the vestibular apparatus by cocaine applications to the middle ear. In 1935 and 1936 it was shown that procaine, hypertonic sodium chloride, diphtheria toxin, quinine and ethyl alcohol and methyl alcohol applied in the middle ear produced marked changes in the vestibular function. It was assumed that the thin and soft fenestra rotunda would be the main route for soluble substances.

Eleven middle ears were used in experiments on dogs to determine the extension of mercurochrome staining in the temporal bones. Photomicrographs presented showed cochleas stained with mercurochrome which had been put into the middle ear cavity. The fenestra rotunda showed heavy stain deposits on the side toward the middle ear but none on the other. The observations in this experiment indicated that

mercurochrome passed through all tissue between the middle and inner ear. The deep staining of the nerve elements could have been accomplished best through penetration of bone. The tissues in decreasing order of definite staining were:

1. Bony wall of the middle ear.

2. Fenestra rotunda.

3. Nerve cells of the spiral ganglion, spiral lamina and VIIIth nerve.

4. Stria vascularis.

5. Articulation of the stapes.

6. Meshes of the aqueduct of the cochlea.

The fenestra rotunda was not the chief transmitter of solutions from the middle to the inner ear.

"Mechanical Aids to Improvement in Hearing" were discussed by Perwitzschky.[69] The author believed that four points should be determined in each individual case before considering the use of mechanical devices for defective hearing:

1. The possibility of improving the hearing.

2. The extent of deafness.

3. The severity of deafness.

4. Bilateral involvement — hearing device should be applied to ear less affected.

Four types of hearing devices were discussed:

A. Tubular devices — ear trumpet or hearing fan devices.

B. Electrical devices: *1.* simple with microphone; *2.* more elaborate with amplifiers.

C. Bone conduction devices of telephone type.

D. Tympanic membrane type.

The author believed it was necessary by careful study to discover the proper device for each individual case.

Predescu-Rion[70] discussed "Hormonal Deafness." In this case the author reviewed various theories concerning the loss

of hearing after the beginning of menstruation and following the first or second pregnancy. Twelve case histories were presented in this discussion, with notes upon parathyroid and thyroid extract treatment.

The author stated that parathyroid extract had been valuable in the treatment of certain diseases such as chronic rheumatism, and one is familiar with deafness in cases of arthritis; that under the influence of ultraviolet rays, ergosterin (found in the skin) was transformed into vitamin B and other substances; that vitamin B had a specific action on calcium fixation; that vitamin D avitaminosis disturbed the calcium-phosphorus equilibrium, resulting in diminished phosphorus absorption. Phosphatemia followed with abnormal proliferation of cartilaginous tissue or bone. Otospongiosis began exactly via these cartilaginous islands around the oval window, by an invasion of round and oval cells such as are also found at a certain stage of fibrous osteitis.

The good results obtained by parathyroid extract in similar types of deafness have shown the complexity of the question.

"The Problem of the Deaf" was presented by Shea.[71] This was a generalized, rather popular review, discussing deafness at different age periods. Emphasis was placed upon the importance of adenoid tissue and the treatment of acute conditions. Many practical points in treatment were included in the paper, with special consideration of the ear of the infant. In the interpretation of the otoscopic picture in infantile mastoiditis, attention was called to the importance of the: *a*. Knowledge of the presence of the notch; *b*. the blending of the tympanic membrane with the periosteum; *c*. the high position of the infantile antrum. The deafness of school children and adults were considered separately.

Eustachian tube pathology was emphasized. Routine inflations and vibrations received comment and the examination for nerve deafness was outlined. Sex elements, dental influence, were briefly analyzed, as well as telephone restrictions. Comments were made on different aids to hearing.

"Sterility and Interruption of Pregnancy in Cases of Otosclerosis" were discussed by Greifenstein.[72] During the author's observation over a period of two years, proposals for sterilization on the basis of health were made in 122

cases. Seventy-nine cases were presented because of well established otosclerosis.

Auditory disturbances were not increased so much in the first pregnancy as in later ones; toxicity and endocrine disturbances were seldom associated with otosclerosis. There has been much diversity of opinion as to a basis for sterilization. In otosclerosis, there is always danger: first that each pregnancy may make the otosclerotic condition worse; and, second, from a eugenic standpoint, there is the probability of transmission to the offspring. The author stated, however, that sufficient research upon this topic had not yet been made.

Jauerneck[73] discussed "Treatment of Acute Middle Ear Suppuration by the Practicing Physician." The author discussed the general symptomatology of acute otitis media and his practice in tympanic incision when that was required. For this procedure, children under age 3 years required no anesthetic; older children, ethyl chloride anesthesia was used, and for adults, local anesthesia. He suggested the following solution as a favorable one for local anesthesia: Pantocain, 1.0; acid. carbol, 1.0; menthol, 2.0; spirit. vin. ad, 10.0. He advised no irrigation after incision, but the protection of the external canal by an ointment.

He advocated consultation with a specialist under the following conditions:

1. Facial paralysis, or symptoms of vertigo. These often denoted serious complications, such as pus in the labyrinth.

2. Pus discharge not responding promptly to treatment, which may indicate a rare form of otitis.

3. Recurrence of fever and pain after an interval of freedom from them, which indicates incipient bone softening.

4. Apparent sinus thrombosis. This was a true sign of meningitis, necessitating immediate lumbar puncture.

5. Persistent otitis and impaired hearing in cases where malignant disease was suspected.

Menck-Thygesen[74] reported on "The Treatment of Acute Otitis Media with X-rays." The author stated that the first actual interest in X-ray treatment in acute inflammation followed favorable results in German research in 1924. This

study was carried on to find out whether the inflammation was checked or only alleviated. The amount of dosage has not yet been universally accepted. The action of the X-rays upon tissues was discussed. Rather than lymphocytic destruction with lowering of tissue tension and subsequent diminution of pain, the author accepted the release of enzymes and antibodies, brought about by autolysis. The author reported X-ray treatment in 110 patients, in connection with other routine care, including paracentesis when necessary. The author concluded that X-ray treatment in acute otitis media proved successful, but this was apparently in combination with other treatment. The following contraindications were stated:

1. Progressive fulminating mastoiditis.

2. Diffuse destruction visible in X-ray studies.

3. Suspected intracranial complications.

Majer[75] discussed "Otitis Associated with Diabetes." The purpose of this paper was to discuss the complicated picture of otitis in connection with diabetes mellitus. In acute otitis complicated by diabetes there was: *1.* marked tendency to extensive bone disturbance, with endocranial complication danger both before and after operation; *2.* general systemic weakness which might lead to coma during the therapeutic period. In 4,500 acute otitis cases, 35 had established diabetes. That is, one out of every 110 otitis cases had diabetes. The tables presented detailed results in the treatment of these 35 cases, 21 of which were operated. The bacteriologic findings in these cases consisted of hemolytic streptococci, streptococcus mucosus and pneumococci. The author emphasized the importance of co-operation between otologist and internist.

"Defective Hearing and Nutrition in Children" was presented by Kerridge, Briggs, Choyce and Hill.[76] This work has added numerical expression to the clinical opinion on the prevalence of middle ear disease among children of the poorer classes. It was apparently from four to 10 times as common in the poorer social conditions.

Climate, housing and the mixing of children made little difference in the incidence of the disease. From the high inci-

dence of defective hearing with deficient diet, probably the most important work on the prevention of deafness must be done upon social conditions, and especially nutrition. No specific food nor vitamin recommendations were made.

Urbantschitsch[77] wrote "Concerning the Question of Shunting Off the Inner Ear in Case of Scarlet Fever." Inflammation of the middle ear and perilabyrinthine suppurations could lead to the cutting off or even destruction of the function of the inner ear. Participation of the labyrinth in the disease process could result in the form of a serous labyrinthitis.

The author reviewed vascular elements of edema, exudate and blood genesis as etiologic factors.

Inflammation of the middle ear and perilabyrinthine suppurations could lead to destruction of the function of the inner ear. This could come about by a serous labyrinthitis. Etiology could have been by blood stream or diffusion of toxins through round and oval windows. Scarlet fever could produce labyrinthitis, destroying the function of the inner ear — always on both sides, and in children quite rapidly. Pathology showed coagulation of endolymph and fibrous thread formation.

"The Hard-of-Hearing Problem in a Student Health Service" was reviewed by Rossman and Hickernell.[78] The present hearing test scientifically made with an audiometer in a soundproof room with a competent operator was contrasted with the old watch tick test for examining groups of college students. The treatment for deafness at that time was to have the student sit in the front row at lectures, use a hearing aid or learn lip-reading. In cases of deficient hearing now, examinations were made by an otologist, and speech analyzed by a psychologist. After customary scientific tests were made, the case was turned over to a competent otologist for treatment.

Chappell[79] discussed "Hearing Defects," enumerating four chief concerns:

1. Consideration of diagnosis and differential diagnosis.

2. The classification — congenital and acquired types.

3. The important requirements for the deafened patients.

4. Necessary occupational change in 40 per cent of deafened adults.

In the third section he cited the important requirements as: *1*. Maintenance of health. *2*. Acquisition of speech-reading. *3*. Vocational readjustment. *4*. Adaptation of proper hearing aid. He considered removal of adenoid tissue the most important single preventive measure.

Gardner[80] discussed "Speech and Hearing Defects." Certain deaf children would not respond spontaneously to normal speech sounds and, hence, could not acquire normal speech through hearing alone. Speech instruction must be supplemented by sight and touch training before they can produce speech sounds, and the sounds then will not be those of normal speech. They must have training in pitch, rhythm, voice quality, articulation and language.

The author discussed loss of hearing at various speech levels as measured by decibels.

Echtermeyer[81] discussed "The Use of Dr. Zajicek's Hormone Ointment in the Treatment of Patients with Defective Hearing." This article reported the visit of Dr. Zajicek, of Vienna, in Berlin for the demonstration of the use of his hormone ointment for the treatment of defective hearing. The author reported three cases, one with otosclerosis and tinnitus aurium, and two with inner ear deafness, who had been noticeably improved after the use of this embrocation. The research technique and exact character of this ointment were not demonstrated.

Feldstein[82] commented on "Excessive Insufflation of Tubes." Following the earlier introduction of insufflation of the Eustachian tube, this treatment became fashionable for all ear disorders, even lesions of the inner ear and auditory nerve, sometimes giving temporary relief, but frequently produced vertigo and other untoward results. Its practice now has been restricted to cases of first degree chronic and subacute catarrhal involvement of the Eustachian tube.

Wullstein[83] reported on the "Practical Value of Roentgen Research on the Labyrinth." The author claimed that Roentgen research on the labyrinth dealt thus far only with the inner ear. In the course of internal otitis, quiescent periods of

the disease occurred though it was still progressive. Such periods could occur during the development of deep-lying foci in the perilabyrinth when objective indications of labyrinthitis were still lacking.

Second quiescent phases followed later if the labyrinthine functions were disturbed in any way. These phases could be of long or short duration. Roentgen rays could play an important rôle in showing the course of illness and disease picture during these significant time lapses. Healing of granulation and sequestration labyrinthitis could be readily established by means of Roentgen rays, and might lead to arrest of the progress of the disease and ultimate disappearance. The author claimed that bone absorption and formation of new bone could progress to very dangerous stages in diseases of the labyrinth were it not for observations made by Roentgen ray.

Under the title, "Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Inner Ear," Shambaugh[84] gave a careful summary on the bibliographic material available in the field of otolaryngology, which included the etiology, pathology and treatment of otosclerosis and labyrinthine deafness of all forms. Eighty-three references were given in the paper.

GROUP 3. MECHANICAL AND SURGICAL PROCEDURES.

Hughson[85] has presented a most careful review of "The Surgical Treatment of Deafness." The one encouraging factor in the otologist's enthusiasm for this new therapy was his appreciation of the ineffectiveness of present efforts. Protection to pioneer surgery of the ear must rest in the thorough training in technique and the selection of appropriate cases for operation. Any departure from normal auditory acuity, whether conductive, neural or combined, should be scrutinized with an eye toward some type of surgical or radical treatment when conservative therapy has failed.

When all conservative measures in elimination of septic foci, with constitutional and general systemic treatment, have proved unavailing, every patient should be prepared to use a hearing aid, whether surgical intervention is contemplated or

not. The psychologic reaction, both to surgical failure and futile treatment, should require careful consideration.

Selection of Cases for Surgical Treatment:

The diagnosis has been simply made up on the basis of a progressive loss of hearing in the third and fourth decade, associated with tinnitus, little involvement of the drumhead, and relatively normal bone conduction; however, the author maintained that recent study indicated conclusively that bone conduction could be impaired by many conditions other than damage to the neural mechanism of the ear. The mobility of the drumhead and patency of the Eustachian tube should be analyzed, and repeated audiograms insisted upon.

In estimating what degree of impairment could be accepted for operation, the author quoted a reported series where 25 dcb. in the critical frequency range seemed to be the maximum gain obtainable with any degree of consistency; therefore, a loss of more than 50 dcb. in the same frequency would seem to make the operation impractical.

The author referred to other important preoperative tests — the loudness balance as a test of differential significance, an estimate of the fixation of the footplate of the stapes, fatigue tests in differentiation between conduction and nerve deafness, thresholds of intelligibility, and, of course, a complete otolaryngological examination and Wassermann test. If the immediate restoration of hearing was essential, the patient should be advised a hearing aid rather than surgical intervention. The influence of heredity has seemed overestimated.

Standards for Operative Procedure:

The author emphasized the importance of technical ability and the accepting of the risk by the patient.

Operations Available at Present:

The author alluded to Barany and Jenkins, Holmgren, Sourdille and Lempert in the staging of an operation for fistulization of the semicircular canal for otosclerosis. The rationale of the procedure was hardly clear, but the fistula must certainly remain open. Lempert's polishing burr or Holmgren's

work on radium, mesothorium or platinum wire might be solving the problem.

In contrast to this difficult though dramatic operation, the operation for fixation of a membrane of the round window with a tissue graft has been relatively simple, and maximum improvement in all frequencies has been as great as in fistulization. Vascularization of the graft took place by the end of the second week and there has been no disturbance of anatomic relations of the middle ear.

The author stated that experience in sectioning the tensor tympani tendon has been entirely disappointing, and there would seem to have been no causative connection between section of the vestibular division of the VIIIth nerve in Ménière's disease and the subsequent improvement in hearing.

The author sponsored with enthusiasm the general principle that in the surgical treatment of deafness lay the future of the otologist's endeavor. Consistent and painstaking follow-up examinations were of utmost importance.

Canfield[56] made a careful report upon labyrinthine fistulas, reviewing the experiments on the "Vital Response to Various Methods of Producing Defects in Bone." Passow suggested a supplementary hole in the otic capsule in cases of chronic otitis or otosclerosis with deafness. An opening was drilled near the footplate of the stapes with striking initial results in the disappearance of tinnitus and improvement of hearing, which was later diminished. Floderus, Matte, Fremel, Bondy and Barany reported similar operations with transient success. Holmgren and Sourdille did many cases but had to reopen the fistulas which regenerated bone had closed.

The physiological reason for improvement in hearing after opening the fistula has not yet been adequately stated. The fact that cholesteatoma will keep fistulas open for years was an intriguing possibility. Whether this was due to pressure, infection or lining of squamous epithelium was uncertain. The question was whether cholesteatoma could be produced by surgical means.

The following methods were tried by Holmgren and his associates:

a. An application of Thiersch skin grafts to fistulas of monkeys was followed by prompt regeneration of the bone and closure of the fistulas as determined by microscopically controlled serial sections.

b. Parietal and visceral autogenous peritoneal grafts laid in carefully with the peritoneal surface and with the fibrous surface against the fistula have been placed. A study of these bones, four to six weeks after the operation, showed evidence of rapid degeneration.

c. Experimental work with metals in contact with bone to prevent regeneration, as suggested by Menegaux and Odiette, was not conclusive.

d. Radiant energy was tried as a force to interfere with cell growth in general. Radium was the element used. It was placed in the fistula for 16 days, and two weeks after that serial sections were made of the bone. No bone regeneration was found but the fistulas were filled with fibrous tissue.

The fistulization operation has certainly been followed by enough improvement to make it worth while in selected cases. The methods of Holmgren and Lempert differed; Holmgren used a scraping gouge and electric drill, Lempert a polishing burr driven by a dental engine. After scraping, the bone was dull; after burring, it was shiny.

The author has done animal experimentation in an effort to answer the questions as to the difference in the effect upon bone of the mechanical processes — the influence of heat and friction of the burr, the drill injury to bone cells and the possible diminution of the bone's power of regeneration. Monkey and cat skulls were used. The scraping and polishing methods were contrasted, and photomicrographs taken of bone regeneration and morphologic variation. He concluded that the response of vital periosteal bone differed significantly when defects were made with different instruments, although the defects were uniformly covered with the same type of tissue. The author assumed that the essential point in maintaining labyrinthine fistulas was the management of the bone itself, with less regard for the nature of the tissue placed over the defects.

"Surgery of Otosclerosis" was discussed by Holmgren.[87] For several years the author had operated on otosclerosis cases with marked improvement in hearing, but after more or less brief periods it was found that the open fistula had healed. A new two-stage operation was attempted:

1. Radical operation was performed with or without transplanted skin, followed by an opening of the fistula.

2. Mastoid processes were opened and the cavity filled with paraffin of 42° C. fusing point. Later, the paraffin was removed, the fistula opened and covered with the thin membrane which had formed about the paraffin.

The article followed the case studies and observations. The author noted that the patient's hearing capacity reached an optimum improvement in one month. The author further noted that audiogram curves were almost identical in cases of large and small fistulas, and concluded that the size of the fistula had little or no influence on the hearing result. Four audiograms accompanied the article.

Campbell[88] in a careful review presented "Experiences with Fistulization of the Labyrinth in Chronic Progressive Deafness." In the selection of patients, three conditions were fundamental:

1. Deafness must be of the conductive type with good bone conduction.

2. Tympanic membrane must be normal.

3. The labyrinth must react normally to caloric tests.

He outlined the following preoperative treatment:

1. Seventy per cent alcohol was instilled in the ear canal four times daily for two days before operation.

2. The head was shaved for two inches around the margin of the auricle the evening before operation.

3. After thorough cleansing with soap and water, and alcohol, sterile dressing was applied.

4. One and one-half hours before operation 4.5 gr. of sodium pentobarbital was given, and hypodermic of 1/6 gr. of

morphine sulfate; 1/150 gr. of scopolamine hydrobromide was administered 45 minutes before operation.

5. Immediately before operation, 70 per cent alcohol was applied to operative area.

In regard to *dangers of the operation,* he maintained that if operation was aseptic and carefully done, there should be no injury to cartilage, labyrinth, facial nerve, tympanic membrane, incus, nor decrease in hearing.

Postoperative reactions and complications might be:

1. Dizziness.

2. Slight pain and temperature.

3. Thick exudative moisture and often troublesome granulation tissue in wound healing.

4. Hearing improvement dependent on maintenance of open fistula and fistula reaction. During first week of convalescence with packing, degree of hearing cannot be determined. Delayed improvement may occur because of tissue reaction. Hearing improvement disappears with closure of fistula and loss of fistula reaction.

The author believed that in properly selected and operated patients, 35 per cent improvement, up to 45 deb. in the lower and middle frequencies, might be expected.

Nager[59] presented a "Demonstration of Otosclerosis Operations on Monkeys, Using the Holmgren Technique." This paper concerned research in regard to the ultimate outcome of the exposed sound fistula in the Holmgren operation.

It was known that fistulization of the semicircular canals generally healed perfectly with bone tissue. Observations were made on cases which healed only with connective tissue, as in cholesteatoma and tuberculosis. Monkeys from India were chosen because of their habitat and conditions of nutrition.

The demonstrations revealed that healing processes were dependent upon different factors, chief of which, in this research, was the extent of time between the operation and death of the animal, and, secondarily, the tissue and substances used in an attempt to keep the sound fistulas open.

There followed certain details on individual animal studies. The author noted that the animals used in this research were not exempt from osteodystrophic disease, as an incision made between the temporomaxillary articulation, whereby the articular cavities as well as the condyles were revealed, showed marked medullary fibrosis and normal bone structures.

While the problem of membranous healing of semicircular canal fistulas had not been solved, some remarkable facts were noted. One was that in only two cases were there signs of labyrinthitis. Placing radium in the wound caused marked retraction in bone formation without injury to the labyrinth. It was also noted that there was almost no evidence of resorption either in old or newly formed bone. The author believed that time alone would demonstrate to what extent mechanical impulses exerted influence in these cases.

The animal experimentation had established the findings which were advanced and demonstrated in recent years by research on otosclerotic temporal bones of human beings. The author believed that further research was warranted as there was no other better outlook for the therapy of otosclerosis. Four illustrations accompanied the article.

Hutchinson[80] discussed "A Case of Membranous Periotic Deafness Treated Surgically by Sourdille's Procedure." After a historical review of the operative treatment of periotic deafness with mention of Kessell, Passow, Barany, Jenkins, Holmgren, Sourdille and Lempert, the author answered three fundamental arguments against operative intervention:

1. In reply to the statement that otosclerosis was a degenerative lesion and surgical measures could lead to no benefit. the author replied that surgery aimed at improving hearing, not curing the disease.

2. In reply to the statement that operation involved risk to hearing and life, the reply was made that hearing was already useless, and operators using aseptic technique have had no mortality.

3. In reply to the argument that bone conduction aids gave similar benefit and without risk, the author replied that periotic deafness was progressive and sooner or later a hearing aid became useless.

The author stated that otosclerosis was a local manifestation of a general disease and suggested two phases of etiology:

1. The presence of constitutional factors, possibly endocrines.

2. The existence of biologically feeble osseous tissue in the region of the anastomosis between vessels of the middle ear and the labyrinthine capsule.

These comments were made on the Lempert technique:

1. The exposure was not as free as a postauricular incision, which made the operation more difficult.

2. The fact that the Eustachian tube was shut off at the same stage as the opening of the semicircular canal would increase the risk of labyrinthine infection.

3. The making of the actual fenestration at the bottom of a concave trough should increase the possibility of what Sourdille calls "decollement" of the flap occurring later.

4. The fenestration was covered by a tympanomeatal flap which had not yet completely cicatrized. This should permit further opportunity of the above "decollement" accident occurring as a result of the contraction of the flap.

Although the title referred to a single case, the writer reported three cases, two of which failed, and the third had regained 60 per cent of normal hearing 23 months after operation.

Careful attention to dental and nasopharyngeal pathology before operation and shutting off the Eustachian tube in the first stage would materially improve prognosis. The author presented three absolute contraindications:

1. Absence of hearing by bone conduction.

2. Presence of otosclerotic change in the bony wall of external semicircular canal.

3. Existence of a low middle fossa and a forward sinus where a free exposure was impossible.

Kopetzky[91] presented "Studies in Labyrinthine Fenestration to Improve Hearing; Preliminary Report." The author stated that the problem of surgical relief for deafness was far

from solution, that surgical work should be continued and physiologists re-examine the fundamentals of hearing. Regarding labyrinthine fenestration, the author agreed with Campbell and Canfield that the tissue destined to cover the fenestration was less important than the manner of making the fistula. The author further believed that the heat which the burr produced was one of the important elements in long inhibiting the closure of the fistula. The size of the fenestration played a part in the end-result. The best results have been obtained with a fenestration of from 1 to 3 mm. in length. The ear first involved in deafness was probably the ear at fault, and he believed that the second ear had lost function largely because there had been a lack of stimulation to the association centres over a long period of time by the initially involved side.

Tumarkin[92] discussed "Surgical Conservation of Hearing." The persistence of chronic perforating epitympanitis with otorrhea but without serious involvement of the drum, ossicles or mastoid has been unnecessarily regarded as an indication for radical mastoidectomy. The alternative transmeatal atticotomy has apparently never been performed in England. This half hour operation under local anesthesia permitted patients to leave the hospital on second or third day. In the presence of gross disease of the ossicles and tympanic membrane, there was nothing to gain from this operation, and it would be contraindicated in chronic suppurative mastoiditis.

The author stated that most cases of chronic otorrhea in adult life and many in childhood were due to epitympanitis, and criticized the frequent failure to distinguish between chronic epitympanitis and chronic panotitis. The author reported no complications in a series of 17 cases.

Padgett[93] reported "An Operation for Possible Alleviation of Certain Cases of Congenital Deafness and Certain Types of Acquired Chronic Deafness." The author first discussed an operation for reconstruction of a new external auditory canal of the postauricular type in congenital malformations with canal atresia. This was suggested because in certain cases of chronic deafness, one could open the Eustachian tube, the ear or the aditus as in a mastoid operation, and while the opening remained, hearing was improved. The operation for a permanent tube by making a new canal post-

auricularly was tried on several patients with chronic deafness and tinnitus. A small rubber tube was inserted, after flaps of skin had been turned under. The stiffness of the tube was used to push the end of the tube flap well within the aditus opening. At a later operation, the new auditory canal could be transplanted to open nearer its normal position. Two-thirds of the 15 cases operated had improved hearing and the annoying tinnitus was completely relieved.

"The Indications for Labyrinth Operation" were discussed by Mayer.[94] After extensive research by the author, the following indications for labyrinth operation were determined:

1. Symptoms of labyrinthine irritation.

2. Deviations in labyrinthine findings.

3. Destruction of the osseous labyrinth wall.

4. Established meningitis.

The analysis of these symptoms were clarified by the author and in conclusion it was emphasized that if these four points were well established, labyrinthine operation was positively indicated.

He discussed the various combinations of these four conditions in their indication for operation. He believed that operation was far less dangerous than postponement in cases of labyrinthine disease.

Sercer[95] described "My First Experience in Operative Therapy in Otosclerosis." All present methods of surgical therapy were based upon either the principles of Wittmaack or Holmgren. The Wittmaack theory was to check the disease by removal of obstruction in the labyrinth. He designated this as "Duralüftung"; that is, circulation of air over the tegmen of the tympani. The theory of Barany, Holmgren and others was to immediately improve hearing by creating a sound fistula at one or more of the semicircular canals. The author then described the technique of making this sound fistula and its covering by gold leaf lamellae and then skin flaps.

The author considered this operation, performed according to the Holmgren method and the Wittmaack procedure equally good, but in his opinion the Wittmaack operation should be reserved for early cases of otosclerosis and the Holmgren

technique for later cases in the hope of gaining at least improvement.

In another medical discussion, not officially reported[96] in article form, it was stated that this operation had been performed 11 times without complications. In all cases improvement of hearing occurred, though in some cases improvement was of short duration. It has, however, persisted in a number of instances for over two years. The maintenance of a permanent, open, sound fistula was, in the author's opinion, a biochemic problem which could be solved only in the selection of appropriate metals for handling the fistula.

Meyer[97] presented "A New View on Deafness in Cases of Otosclerosis and a Possible New Method for Treatment; Part I." Ancient and modern authors emphasized the difficulty of making a certain diagnosis of otosclerosis, rather than simply stapes ankylosis. The operative treatments of Holmgren, Sourdille and Wittmaack were complicated and not yet assured of success. In view of the assertion of Holmgren that decompression of the peri- and endolymph should improve hearing, the author sought to find how decompression of the labyrinth fluid would influence hearing in otosclerosis. The preliminary success of suboccipital puncture indicated its feasible acceptance for certain cases. Two cases were reported out of a total of six. Results in these were apparently favorable.

SUMMARY.

1. Six cases of deafness, not caused by any inflammation, were presented. Four of them, which were typical cases of otosclerosis, were treated by suboccipital puncture with release of 30 to 55 cc. of cerebrospinal fluid.

2. The influence on hearing and tinnitus was favorable in all cases.

3. From a scientific standpoint, reduction of fluid pressure improved hearing and noises.

4. Hearing improvement was sometimes astonishing, even up to normal.

5. It was found that the upper sound limit was not a sure sign of labyrinth atrophy.

Meyer,[98] in "A New View on Deafness in Cases of Otosclerosis and a Possible New Method for Treatment; Part II," presented this thesis:

1. Typical hardness-of-hearing in case of otosclerosis is not stipulated by ankylosis of the stapes alone.

2. The upper tone limitation by air conduction in advanced otosclerosis is not a proof of secondary labyrinthine atrophy.

Not only the open artificial labyrinthine fissure but also decompression of the labyrinthine fluid is of importance for improved hearing. A suboccipital puncture which decreased pressure without a complicated operation had proved successful in six cases.

The author reported that in a series of 12 cases, only one was unsuccessful, and this was because of otosclerotic changes in the neighborhood of the round window. The author further discussed the upper tone findings by air and bone conduction.

Passe[99] reported "Surgical Treatment of 14 Cases of Otosclerosis." In this series, 12 cases were done under the Holmgren and two under the Lempert technique. The majority of the cases were well advanced, all had family history of deafness, and the more deafened ear was selected for operation. Tinnitus was always present.

Frequent audiograms were taken and the operations were done under local anesthesia.

After operation, hearing was improved in five cases according to the audiometer, only three improved to the extent of aiding conversation, but the other two were able to use a hearing aid with benefit. Tinnitus disappeared in two cases and greatly improved in seven.

In one case, although the fistula sign was present, hearing was decreased.

The author suggested that if a new blood supply to the saccus endolymphaticus could be established, a membranous covering to the fistula would suffice.

"Consideration of the Surgical Treatment of Dry Otitis" was presented by Hubin.[100] This paper was principally con-

cerned in the symptomatology of otosclerosis, the explanation of the hearing through the third window, and the operative technique described by Sourdille, with its three stages. One progressive case of this form of deafness was presented by the author, with brief account of examination, operation and postoperative events. This was considered a favorable case.

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STENOSIS OF THE LARYNX DUE TO PARALYSIS OF THE VOCAL CORDS. TREATMENT BY SUBMUCOUS RESECTION OF THE VOCAL CORDS. REPORT OF OPERATED CASES.*

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Stenosis of the larynx due to paralysis of the vocal cords has long challenged the surgical ingenuity of laryngologists. The time-honored operation of tracheotomy, with its numerous technical variations, has been replaced at intervals by some plastic procedure designed to produce a more satisfactory end-result for the patient. A continuous effort has been made to achieve the two prime requisites for a thoroughly satisfactory relief from this condition. A retention of the natural airway and preservation of the voice have been one or both objectives dominating the minds of those who have proposed new methods of treatment.

Not all cases of bilateral paralysis of the vocal cords require the surgical re-establishment of an airway. Nor is aphonia the inevitable symptom of such paralysis. The disability produced by such paralyses depends upon the position assumed by the paralyzed cord. Whether or not the law of Semon is valid and immutable, and whether or not a vocal cord paralyzed by injury to the recurrent laryngeal nerve comes immediately to lie in the paramedian or the cadaveric position is not the urgent concern of the laryngologist whose responsibility is the maintenance of an adequate airway in a patient who is dyspneic from laryngeal stenosis. It is of great importance, however, for one to realize that the position of a paralyzed vocal cord due to recurrent laryngeal nerve injury may change, as suggested by New.[1] This likelihood of shift of a vocal cord from the cadaveric to the median position following an insult to the recurrent nerve prompts the laryngologist to keep under closer observation the individual who is moderately inconvenienced with both cords fixed in the cadaveric position. Because of the possibility of these cords returning

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to the median line, the patient is a potential candidate for surgical relief from an impending dyspnea.

Although it is not the purpose of this paper to deal with the possible etiologic factors concerned in larnyngeal paralysis, it is important that the laryngologist acquaint himself with this subject. A careful history, a thorough medical and

Fig. 1. The two way tracheotomy tube which has been used for continuous dilatation in cases of laryngeal stenosis of various types consists of a curved tracheal cannula inserted through a window in a rubber tube. The size of the cannula and rubber tubing can be varied according to the size required. The advantages in this type of two-way tube lie in the minimal reaction produced within the larynx and the ease with which it can be removed. Occasionally it is necessary to enlarge the tracheotomy wound to facilitate extraction but in most instances the flexibility of the tube at the level of the window permits of easy removal.

neurological examination and an intelligent interpretation of the laryngeal findings will serve in the majority of cases to determine the etiologic background of the disorder and to suggest the most appropriate procedures for relief. The surgical therapy of the condition must be individualized, for it can scarcely be said that every patient with bilateral paralysis of the vocal cords can be adapted to one routine standard surgical operation.

Felix Semon,[2] in 1879, advocated careful observation of the patient with bilateral paralysis of the posterior cricoarytenoid muscles and tracheotomy for the relief of the dyspnea. Sir St. Clair Thomson,[3] in 1931, continued to support tracheotomy as the treatment of choice in this affliction. He stated that there was no danger in inspiring unwarmed air into the trachea, particularly in the case of the adult, in whom the value of nasal respiration is less great than in children. He stated that social disability scarcely exists for the tracheotomized patient because the tube may be concealed easily, and with a speaking attachment to the tracheotomy tube, a fair voice can be maintained. Sir St. Clair Thomson also contended that there was no special danger of bronchitis in the tracheoto mized patient, for when catarrh does occur, its development is due to a high position of the tracheotomy tube which enters the trachea through the narrow, vascular, glandular and sensitive subglottic larynx. That permanent cannulization is the treatment of choice is also suggested by Negus,[4] who has advocated the use of a butterfly valve in the mouth of the tracheotomy tube which will permit free inspiration of air and allow expiration through the glottis, thus maintaining the voice.

John E. MacKenty,[5] in 1928, developed an operation for the relief of abductor paralysis of the larynx, characterized by the formation of a permanent fistula in the neck, produced by the removal of an oval section of tracheal rings and the suture of the tracheal mucous membrane to the skin of the neck. The object of this variation in the technique of tracheotomy was to form an adequate permanent airway which could be maintained without the wearing of a tracheal cannula.

In an effort to reconstruct an airway at the possible expense of the voice, numerous laryngeal plastic reconstructive operations have been devised. Some years ago it was thought that the ventricle stripping operation, long successful on the horse disabled by laryngeal paralysis, might be applicable to man. Michael Vlasto,[6] in 1921, following operations on the cadaver, concluded that, theoretically at least, the operation was not suited to the human larynx. He stated that the ventricle and saccule do not come down over the lateral surface of the true cord as they do in the horse and stripping would not bring the cord out into a lateral position. A modification of this

technique was devised by Chevalier Jackson,[7] who used it in a number of cases with some degree of success. The operation was called the ventriculocordectomy, and consisted essentially of removing the vocal cord and adjacent ventricular floor by means of a punch forceps, which resulted in a laterally displaced cicatricial contracture, allowing for an increased airway. Wittmaack[8] deviated from the previous course of striving to establish a horizontal laryngeal slit and attempted to displace one vocal cord downward. His operation consisted of a laryngofissure and the removal of an arytenoid cartilage with its process muscularis through a mucous membrane incision. Following the suture of the mucous membrane incision, the posterior end of the vocal cord was displaced downward by scar tissue contraction, thus eliminating the stenosis which had been produced by approximation of the two cords. Loré,[9] in 1936, presented a preliminary report on an operation for the relief of double abductor paralysis, in which there was seen another attempt to produce lateral displacement of the vocal cords. Proceeding on the premise that a vocal cord is held straight by two points, the anterior commissure and the vocal process of the arytenoid, and that lateral displacement of one fixed point should serve to increase the usable airway, he exposed the larynx by a thyrotomy and plunged a sharp scalpel into the floor of the ventricle through the submucosal tissue of the vocal cord and out into the subglottic space below. The knife was inserted at the anterior end of the vocal cord and carried posteriorly, cutting through the tip of the vocal process and proceeding to the interarytenoid space, where the mucous membrane was severed. Following this procedure, the arytenoid was removed, as was the tissue subjacent to the detached cord. After this removal of tissue, the vocal cord was replaced, anchored by sutures, the larynx packed with gauze or a rubber balloon, and the laryngofissure closed. This was, in effect, the removal of the arytenoid cartilage and vocalis muscle by an intralaryngeal approach.

Another method of lateral displacement of the vocal cords was described by Rethi,[10] who divided the adductor muscles and held the arytenoids in lateral position by tampon until ankylosis of the cricoarytenoid joint developed.

In 1932, W. B. Hoover[11] reported his results on the operative treatment of bilateral abductor paralysis of the larynx by

submucous resection of the vocal cords. In the operation which he describes, a tracheotomy is done, following which a laryngofissure exposes the interior of the larynx. A submucosal resection of the muscle and fibrous cord is then accomplished, including the removal of the vocal process. The mucous membrane of the larynx is held against the thyroid cartilage by a pack brought out through the cricothyroid membrane, then the cartilage, subcutaneous tissue and skin are sutured together.

The various procedures above described scarcely satisfy the prime requisites of a thoroughly satisfactory result. The desired preservation of voice and natural airway practically demands restoration of function of the vocal cords. Since nerve anastomoses have brought about the return of function of other muscles, notably those of the face following substitution anastomotic operations on the facial nerve, efforts to reproduce these results within the larynx have been tried from time to time. E. Broughton Barnes and Sir Charles Ballance,[12] in 1927, reported a case in which anastomosis between the recurrent laryngeal and phrenic nerves was performed in an effort to restore the function of paralyzed vocal cords. On one side, this anastomosis was accomplished by splitting the phrenic nerve and joining a portion of this nerve end to end with the severed recurrent laryngeal. On the other side, an end to side anastomosis was done, in which the severed recurrent laryngeal nerve was implanted into the side of the intact phrenic nerve. Temporary improvement was observed on one side and apparently permanent improvement noted on the other, that in which an end to end anastomosis had been used. In 1924, Charles H. Frazier[13] reported in the *Annals of Surgery*, and later in the *Journal of the American Medical Association*, cases of recurrent laryngeal nerve paralysis treated by substitution anastomosis operations in which the ramus descendens hypoglossi was utilized. Although encouraging early results were obtained, the attempts were not uniformly successful and this type of procedure can scarcely be said to be adaptable to many cases. It must be admitted, however, that its successful accomplishment would produce the ideal result.

In the past 10 years at the University of Michigan Hospital clinic, there have been recorded 29 cases of bilateral abductor

paralysis of the larynx. There were nine more on the basis of traumatic, inflammatory and neighborhood neoplastic conditions not included in this group, which comprises only those paralyses of neuropathic etiology. It was found that the largest number of cases was associated with thyroidectomy. These figures corroborate the statistics from various other clinics. It was enlightening to learn that vocal cord paralysis resulting from an enlarged nonmalignant thyroid was a rare occurrence. In this series there were two instances in which an incomplete paralysis was associated with thyroid enlargement. In nearly 50 per cent of these cases the paralysis of the vocal cords developed on the basis of a central lesion.

TWENTY-NINE CASES OF BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX.

No.	ETIOLOGY	PARALYSIS		TREATMENT			
		Complete	Incomplete	None	Permanent Tracheostomy	Temporary Tracheostomy (Recov.)	Submucous Resection Vocal Cords
14	Postoperative thyroid	11	3	6	2	3	3
4	CNS lues	1	3	3	----	----	1
3	Cause unknown	1	2	2	----	----	1
2	Bulbar palsy	----	2	2	----	----	----
2	Enlarged thyroid	----	2	2	----	----	----
1	Syringobulbia	1	----	----	1	----	----
1	Cerebellar syndrome (degenerative)	1	----	1	----	----	----
1	? Arteriosclerosis	----	1	1	----	----	----
1	? Amyotrophic lateral sclerosis	----	1	1	----	----	----

In the preceding table, the type of treatment instituted in each of the 29 cases in this series is outlined. The majority of these patients had incomplete paralysis or apparently complete paralysis, with the vocal cords lying in the cadaveric position, so that an adequate minimum airway was maintained and surgical treatment not required. In four post-thyroidectomy cases the paralysis was temporary. In three instances of post-thyroidectomy bilateral recurrent laryngeal nerve paralysis, one patient had an adequate minimum airway for nine

months, another for six years, and yet another for 17 years before developing an embarrassing dyspnea. At the time of the examination of each of these patients in this clinic, both cords were fixed in the midline, which situation was apparently of recent development, bearing out the theory of New and Childrey[1] that paralyzed vocal cords, associated with recurrent nerve injury come eventually to lie in the midline.

In five instances of laryngeal stenosis associated with bilateral abductor paralysis of the vocal cords, a submucous resection of the larynx was performed. The operation used in four of the cases varied in technique from that described by Hoover in two essentials: *1.* the arytenoid cartilage was completely removed along with the process muscularis in an effort to widen the glottic chink; and *2.* a two-way tube was placed in the larynx following the laryngofissure and left in place from one to three months.

REPORT OF CASES.

Case 1: L. H., 300,570, female, age 56 years. This patient was in good health until approximately five years before coming to the clinic, when she began to notice weakness, insomnia and nervousness; also suffering occasional knife-like pains of a girdle character. During the past year the patient experienced fainting spells, difficulty in writing, change of pitch in the voice and increasing dyspnea. Examination revealed evidence of a central nervous system lues associated with bilateral abductor paralysis of the larynx. She was extremely dyspneic, even at rest. A tracheotomy was done, followed in five days by a submucous resection of both vocal cords under ether anesthesia. The resection included the vocal processes but not the arytenoid cartilages. The cricoid cartilage was split in the midline and a two-way dilating tracheotomy tube inserted and left in place for one month. Following the removal of this tube, the patient maintained an excellent airway and developed a hoarse but serviceable voice.

Case 2: V. M., 401,708, female, age 29 years. This patient entered the clinic with a complaint of progressive hoarseness and dyspnea of three years' duration. A thyroidectomy had been done without relief from symptoms. Examination revealed a bilateral abductor paralysis with minimal airway.

Neurological and general physical examination, blood studies, radiography of the chest and neck failed to reveal the cause of the laryngeal paralysis. Under ether anesthesia, a submucous resection of both vocal cords was performed. Both arytenoids were removed at this operation and a gauze pack was inserted into the larynx to hold the mucous membrane against the thyroid cartilage. A tracheotomy tube was placed in the low position. At the time of the operation it was noted that the larynx was exceedingly small, and doubt was expressed that a submucous resection of the vocal cords would result in an adequate airway. The gauze pack was removed from the larynx after 48 hours and for a time the result seemed to be satisfactory, but within three weeks the patient returned with larnygeal obstruction due to a mass of granulation tissue occluding the glottic chink. A two-way dilating tracheotomy tube was inserted for three months, resulting in an adequate airway temporarily. A Jackson one-way tracheotomy tube was left in place during a period of observation, and after eight months the larynx again filled with granulation tissue. It was advised that a two-way dilating tracheotomy tube be again inserted but the patient has not returned for this procedure during the year since it was advised.

Case 3: R. N., 433,577, female, age 39 years. This patient gave a history of bilateral thyroidectomy seven years before coming to the clinic. This was followed by a persistent, moderate dyspnea and hoarseness. Seven months before admission the dyspnea became acute and demanded tracheotomy. The cannula had been retained since the tracheotomy was performed. Examination revealed the right vocal cord to be fixed in the midline and there was but very weak abduction on the left side. Under local anesthesia, a submucous resection of the right vocal cord, including the entire arytenoid, was done. A two-way dilating tracheotomy tube was inserted into the larynx and allowed to remain for three months. After its removal, a one-way tube was left in place for one week during a period of observation. The end-result was an excellent airway and a serviceable voice.

Case 4: R. D., 427,412, female, age 24 years. This patient gave a history of having undergone a thyroidectomy 11 months prior to admission to the clinic, and, immediately following, some dyspnea and hoarseness developed. The difficulty in

breathing gradually increased until two months before entering here, when it was necessary to perform a tracheotomy. At the time of examination in the clinic, the tracheotomy tube was used as an auxiliary airway and the patient was able to talk with a husky voice. Laryngoscopy revealed fixation of both vocal cords with a very small glottic chink. Under local anesthesia, a submucous resection of both vocal cords, including both arytenoids, was performed. A two-way dilating tracheotomy tube was then inserted and left in place for three months, when it was replaced by a Jackson one-way tracheotomy cannula, which was to be used during a period of observation. It was necessary on two occasions to remove granulation tissue from the larynx but after a period of nine months the patient had an excellent airway. The voice was very hoarse, but no worse than before the resection of the vocal cords.

Case 5: L. E., 415365, female, age 66 years. This patient gave a history of having had a thyroidectomy performed 17 years before, which was immediately followed by dyspnea and hoarseness, which had persisted. Four weeks prior to admission to the clinic, the dyspnea became acute. At the time of examination the patient's respirations were greatly embarrassed and the larynx demonstrated a bilateral abductor paralysis of the vocal cords with fixation in the midline. Under local anesthesia, a submucous resection of both vocal cords, including the arytenoids, was done without incident. A two-way dilating tracheotomy tube was inserted into the larynx and left in place for two months. Following the removal of this tube, the airway remained adequate. The voice was poor but serviceable.

TECHNIQUE OF THE OPERATION.

Although a submucous resection of the vocal cords may be done under either general or local anesthesia, the latter possesses some advantages. The position of the patient should be the same as for a tracheotomy, with a pad under the shoulders and the head well back, with the neck extended. A midline incision is made from the level of the hyoid bone to a point about 1 cm. below the cricoid cartilage. The ribbon muscles are separated in the midline and the thyroid and cricoid cartilages and upper two rings of the trachea are exposed. The larynx is then split in the midline, exposing its interior. With

a subperiosteal elevator the mucoperichondrium is separated from the internal aspect of the thyroid cartilage. With the No. 15 Bard-Parker knife, the vocalis muscle and vocal cord are dissected free from the mucous membrane of the larynx and removed with a punch forceps. The procedure is facilitated if the tendinous attachment of the vocalis muscle to the vocal process of the arytenoid is exposed and severed. With a small subperiosteal elevator, the arytenoid cartilage may be freed and delivered in one piece. It is desirable to avoid buttonholing the mucous membrane over the vocal cord, as such a tear might encourage the growth of granulation tissue in the interior of the larynx. Following this procedure on one or both sides of the larynx, as the condition warrants, the cricoid cartilage and first ring of the trachea are split in the midline and a two-way tracheotomy tube of the type shown in Fig. 1. is inserted into the larynx. It is important to have the upper dilating end of this two-way tube projecting but slightly over the edges of the vocal cords superiorly. After the insertion of this tube, the edges of the thyroid and cricoid cartilages are sutured together. The soft tissues above and below the tracheotomy tube are then united.

This two-way dilating tracheotomy tube should be left in place for two to three months, during which time healing takes place and the mucous membrane of the larynx becomes adherent to the thyroid cartilage, increasing the size of the airway.

SUMMARY.

A brief review of some of the methods of surgical treatment of laryngeal stenosis associated with bilateral paralysis of the vocal cords is presented.

Twenty-nine cases of bilateral paralysis of the vocal cords seen in the University of Michigan Hospital in the past 10 years are reported and analyzed regarding etiology and treatment.

Five cases subjected to submucous vocal cord resection by modifications of the technique proposed by Hoover are recorded.

CONCLUSIONS.

In selected cases, a submucous resection of the vocal cords offers satisfactory relief from the dyspnea produced by bilateral abductor paralysis. It falls short of producing an ideal end-result, however, in that there is in most instances an unsatisfactory voice.

The objection that food and fluids may flow into the trachea because of the loss of the protective sphincter mechanism of the larynx has not been encountered in the operated cases presented.

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PATHOLOGIC CONDITIONS OF THE ESOPHAGUS.*

DR. LOUIS H. CLERF, Philadelphia.

The esophagus and its diseases receive scant consideration in undergraduate medical instruction. Certain conditions, as cicatricial stenosis, cancer of the esophagus and pulsion diverticulum of the pharynx, are well understood; however, patients complaining of indefinite subjective sensations which cannot be described or localized except in a very vague manner frequently are considered as hysterical or neurotic. These usually are referred from one physician or out-patient department to another, and much valuable time often is consumed before the case is brought to a final conclusion. While many otolaryngologists do not perform esophagoscopy, it, nevertheless, is important that they acquaint themselves with diseases of the upper esophagus and hypopharynx, also with those pharyngeal lesions which manifest symptoms referable to the esophagus.

Before proceeding with a consideration of some of the esophageal diseases which may be met with in general otolaryngologic practice, certain symptoms and diagnostic procedures should be discussed. All diseases of the esophagus manifest themselves sooner or later by the common symptom of difficulty in swallowing — dysphagia. Preceding this, vague, indefinite sensations are described which often cannot be localized. While there may be no objective organic basis for these, it is important to carry out appropriate investigations. Patients with cancer of the esophagus have been observed who complained only of a lump in the throat, and the true nature of the disease was not suspected until food lodged proximal to the growth, producing complete obstruction.

Painful swallowing is observed in periesophageal and hypopharyngeal inflammation and in foreign bodies, particularly if pointed or irregular.

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Cough may be a symptom of esophageal disease and usually results from overflow of mouth secretions or foods, particularly liquids, during attempts at swallowing. It also may occur in esophagotracheal fistula.

Hoarseness, the result of paralysis of a vocal cord, may occur in carcinoma of the cervical esophagus.

Regurgitation is a common symptom of obstructive disease, particularly of the thoracic portion of the esophagus. Tolerance to retained secretions and foods is soon established and, if long continued, dilatation of the esophagus results.

While the diagnosis of many conditions producing esophageal symptoms is a general medical problem, there are two objective diagnostic methods which should be resorted to in all cases; namely, Roentgen ray study and esophagoscopy.

The Roentgen ray examination consists of fluoroscopy and radiography, first without a radiopaque mixture to determine the presence of visceral disease, then with a swallowed opaque mixture to outline the walls of the esophagus. In certain cases a barium-filled gelatin capsule should be employed. The employment of a barium mixture is indispensable and should be used in infants as well as in adults.

Esophagoscopy affords all the certainty of objective information and histologic examination of any removed specimens of tissue. It should be employed in every case of suspected esophageal disease with disturbances in the swallowing function. This procedure is safe in the hands of one properly trained. Blind bouginage is dangerous and inconclusive.

Dysphagia may be the first and often the only symptom of disease of certain periesophageal structures. Aneurysm of the aortic arch, carcinoma of the upper lobe of a lung, malignant metastasis to mediastinal lymph nodes or primary neoplasm of the mediastinum may produce compression stenosis of the esophagus or may involve a recurrent laryngeal nerve, particularly the left, and produce paralysis of the larynx. It is important, therefore, that the otolaryngologist should examine carefully the upper air and food passages in all of these cases. Not infrequently, diseases of the fauces and pharynx manifest themselves by vague sensations referable to swallowing. Among these are hypertrophy or inflammation of the lingual

tonsil, follicular pharyngitis, pharyngeal and hypopharyngeal tumors, particularly carcinoma, and retropharyngeal cellulitis. Not infrequently, patients complaining of a sticking pain or a sensation of a lump in the throat attribute this to swallowing a fish bone, toothpick, match stick or toothbrush bristle. On further questioning, the foreign body history is indefinite, there is no difficulty when foods are swallowed, and the sensations are noted only when swallowing saliva. A careful search of the mouth, throat, pharynx and larynx should be made not only for foreign body or new growth but also for evidence of inflammation of the lymphoid tissues in these areas.

Palpation of the neck should be a routine procedure and should include a search for tender areas, tissue fixation and palpable masses, as enlarged lymph nodes. Points of reference of pain are misleading. It is well known that pain occurring in inflammation or ulceration of the posterior part of the larynx may be referred to the ear; a foreign body in the esophagus at the suprasternal notch may be localized in the lower chest. So, also, the patient with a localized abscess of the lingual tonsil may localize his complaints in the region of the cricoid cartilage.

Symptoms referable to the swallowing function may be local manifestations of a systemic condition. This is well demonstrated in the syndrome described by Plummer and Vinson, which bears their name. While the subjective sensation of a lump in the throat and dysphagia are localized to the cricopharyngeal region, the objective findings of fissuring at the angles of the mouth, atrophy of the mucosa of the tongue, mouth and pharynx, and anemia should immediately suggest the need for medical consultation in addition to a Roentgen study of the swallowing function. General medical examination and appropriate blood studies will give a clue to the underlying disease. Patients suffering from hypothyroidism may exhibit none of its objective evidence. The subjective symptoms often are very indefinite and may be referred to the throat, neck, shoulders or chest, and often are associated with disturbances in swallowing, although there is no Roentgen evidence of esophageal disease.

In the newborn, the occurrence of choking attacks and cyanosis when fluids are taken should suggest atresia of

the esophagus. The respiratory symptoms, which often are alarming, commonly direct attention to the airway, notably the larynx, and very often a provisional diagnosis of congenital laryngeal stenosis is made. In every infant with choking attacks, a study of the swallowing function, using a small quantity of barium mixture, should be carried out (see Fig. 1).

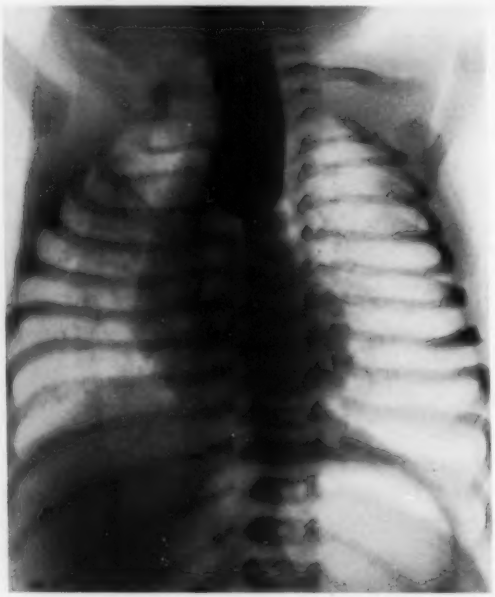

Fig. 1. Roentgenogram of an infant, age 5 days, with congenital atresia of the esophagus. The symptoms were frequent attacks of choking with cyanosis, particularly when fluids were given. Aspiration of mouth secretions gave temporary relief. Thymic enlargement was suspected and two Roentgen treatments were given without benefit. Laryngeal stenosis was suspected, but before proceeding with direct laryngoscopy, functional study of the esophagus was recommended. The diagnosis was obvious.

Although not considered a part of the food passages, the larynx participates in the act of deglutition, and in the presence of certain pathological conditions, disturbances of swallowing may be a prominent symptom. It is not uncommon to have patients with unilateral complete paralysis of the larynx complain of difficulty with swallowing liquids, particularly water, and frequently choke while eating. Paralysis of the

larynx following thyroidectomy may be associated with troublesome dysphagia.

Cricopharyngeal carcinoma constitutes one of the most difficult problems in diagnosis. Objective obstruction to the passing of foods is a late manifestation. The early symptoms are indefinite, and hysteria often is considered as a diagnostic possibility. The diagnosis can be made early only by recognizing the vague sensations, as possible manifestations of cancer, and by esophagoscopic examination. Careful examination by mirror laryngoscopy of the posterior part of the larynx, particularly the arytenoids, for evidence of slight inflammation or fullness and suggestive impairment of motility, and the presence of secretions in one or both pyriform sinuses should be a routine procedure. Palpation of the neck, particularly along the posterior border of the ala of the thyroid cartilage and the signet of the cricoid, may reveal a sensation of induration or fixation of the tissues; slight tenderness may be elicited.

In pulsion diverticulum of the pharynx, gurgling noises in the throat when swallowing foods or mouth secretions, the occurrence of cough, particularly during sleep, and finding food particles in the mouth when assuming a recumbent position often are complained of (see Fig. 2). Eliciting a gurgling sound by compressing the root of the neck immediately after swallowing mouth secretions (Boyce's sign) and observing frothy secretions in a pyriform sinus by mirror laryngoscopy are very suggestive of pharyngeal diverticulum. These signs may occasionally be noted in cases of cancer or stricture of the cervical esophagus, particularly if there is dilatation of the esophagus proximal to the stenosis. Roentgen study and esophagoscopy are conclusive (see Fig. 3).

The treatment of pulsion diverticulum is surgical extirpation of the pouch and repair of the pharyngeal wall. Bouginage not only is dangerous but is worthless, as there is no organic stenosis present. Compressive stenosis of the esophagus occurs when the pouch is filled, and this interferes with swallowing.

Cricopharyngeal spasm often is designated as "globus hystericus" and considered as a manifestation of hysteria. Cricopharyngeal spasmodic stenosis is essentially a spasm of the

horizontal fibres of the inferior constrictor muscle of the pharynx, the cricopharyngeus. The chief complaint is difficulty in starting a bolus of food downward, and associated with this may be a sensation of a lump in the throat which moves upward and downward with swallowing. Spasm may be due to organic disease of the esophagus or to disease of remote organs. A patient was recently observed who had difficulty starting the bolus of food. Cancer of the lower end of the esophagus was found by esophagoscopy.

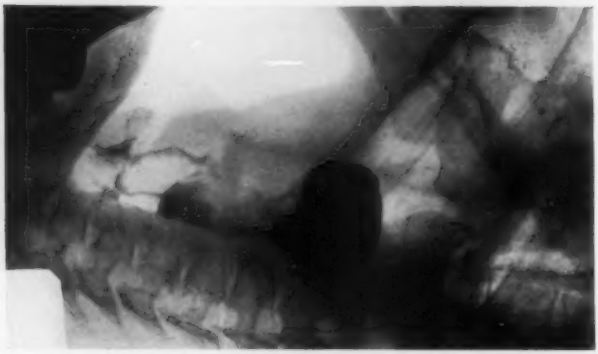

Fig. 2. Roentgenogram made in the lateral plane after swallowing barium while dorsally recumbent exhibits a large collection of the material in the pouch lying behind the esophagus. This pouch communicates with the pharynx and its contents gravitate upward as soon as the pharynx becomes dependent. There is no sphincter surrounding the neck of the pouch. During sleep, with the head low, the contents of the pouch, gravitating into the pharynx, often are aspirated into the trachea.

Roentgen ray study is indicated to determine if stenosis is present. Esophagoscopy should be performed to decide whether the stenosis is spasmodic, organic or both.

Cardiospasm, designated by many terms and ascribed to various causes, is characterized by marked dilatation of the thoracic esophagus, chronic esophagitis and retention of large quantities of food and mouth secretions. The symptoms commonly noted are a feeling of fullness or discomfort referable to the mediastinum, foul breath, an unpleasant taste, with frequent expectoration and regurgitation of stale food. There may be weight loss. Regurgitation may occur while eating or may be deferred several hours. The quantity varies from

several mouthfuls to several pints. Actual pain usually does not occur. The vague discomfort may be relieved by regurgitation or by drinking a glassful of water and forcing the contents of the esophagus into the stomach.

The diagnosis of cardiospasm requires the exclusion of organic disease of the esophagus. It may be simulated by cancer of the cardiac end of the stomach with extension to the

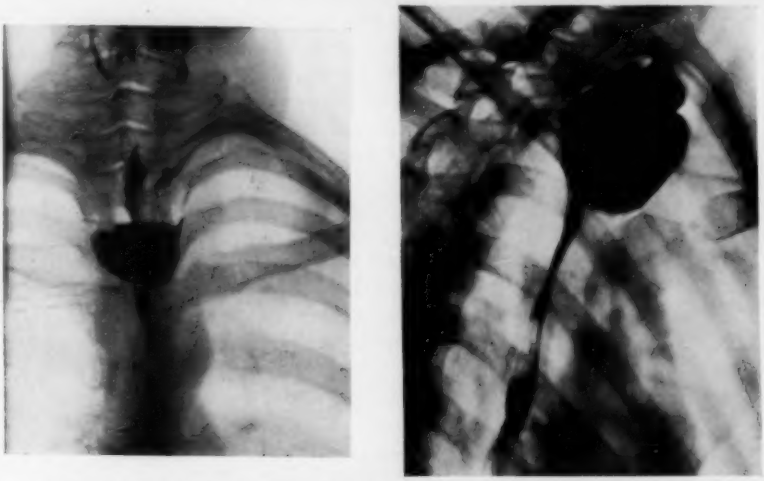

Fig. 3. Roentgenograms made in the P-A and lateral planes in a case of cicatricial stenosis of the esophagus. There is a history of swallowing lye when a child. The case was considered as one of pulsion diverticulum of the pharynx. The lateral Roentgenogram shows the esophagus coming off the fundus of the pouch instead of being anterior to it, as in the case of diverticulum. By esophagoscopy the true nature of the condition was clearly demonstrated.

esophagus. By Reontgen study a correct diagnosis can usually be made. Esophagoscopy should be done, however, to confirm the diagnosis and to exclude malignancy. Treatment consists of divulsion of the hiatus esophageus. The diet should be investigated. General medical studies are indicated in a search for a possible etiologic factor. Relapses occur in about 20 per cent of cases and additional dilatations are required.

Paralysis of the esophagus may be motor or sensory. The causes may be toxic as in diphtheria, functional as in hysteria,

peripheral resulting from neuritis, and central or bulbar as in glossolabiopharyngeal paralysis. The only symptom referable to the esophagus is difficulty or inability to swallow. By mirror laryngoscopy the pyriform sinus will be seen filled with secretions. Overflow of secretions into the larynx with the occurrence of respiratory symptoms are common.

There is a constant tendency to mistake esophageal paralysis for hysteria. Paralysis is practically always associated with other paralyses about the upper food and air passages and are easily seen if looked for. In all cases of inability to swallow, particularly if it is referred to the upper esophagus, the larynx should be observed during the act of swallowing. In organic stenosis, even atresia, there will be elevation of the larynx during the act of deglutition. In paralysis, this movement will be feeble or absent.

A young woman was recently observed who had choked while swallowing and was believed to have a bolus of food obstructing the esophagus. She was febrile and appeared ill. There was present paralysis of the soft palate. The pyriform sinuses were filled with secretions. On attempted deglutition there was no upward movement of the esophagus. The medical consultant made a diagnosis of acute anterior poliomyelitis, which was corroborated by the subsequent clinical course of the disease.

Foreign Bodies in the Esophagus: The occurrence of dysphagia, a sticking sensation when swallowing or pain on slight pressure over the trachea following an attack of choking or gagging while eating or while holding something in the mouth should immediately suggest foreign body in the laryngopharynx or esophagus. Any digital or blind attempt at investigation is absolutely contraindicated. The passage of a bougie is dangerous. Attempts to dislodge the object by swallowing bread crusts, cotton or other materials is hazardous and unscientific. It should be borne in mind that any attempt to force onward in the esophagus an irregular, jagged-edged or pointed object invites trauma, even fatal perforation of the esophageal wall.

A Roentgen study should be made for purposes of diagnosis and localization. This should be performed without and if

necessary with an opaque mixture. Esophagoscopic removal is the only method that is safe, scientific and effective.

In closing, additional emphasis should be placed on the importance of taking a sympathetic attitude towards the patient who complains of esophageal symptoms, irrespective of their apparently trivial or absurd character. Careful and complete examination of the upper air and food passages should be made. Palpation of the neck should be a routine procedure. Roentgen study of the swallowing function should be done if the symptoms or signs cannot be satisfactorily explained by physical examination. Esophagoscopy is indicated in all cases of esophageal disease requiring corroboration of the Roentgen findings. It also is indicated in those cases with esophageal symptoms and in whom there is reasonable belief that disease of the esophagus exists in the presence of negative Roentgen findings. It is important to bear in mind that in the practice of medicine errors by omission are more common than errors by commission.

1530 Locust Street.

HOARSENESS — A NEW CLASSIFICATION AND A BRIEF REPORT OF FOUR INTERESTING CASES.

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It is my intention primarily in this communication to present a systematized classification of the causes for hoarseness, to demonstrate the frequency with which this symptom is encountered in various disease entities and to briefly report a few cases that are of interest from a diagnostic standpoint.

Hoarseness may be defined as any alteration in the speaking voice which results in a roughened or rasping character to the voice. To properly understand such alteration, one must have a clear conception of the mechanics of voice production.

Voice is produced in the larynx by vibrations of the vocal cords, which are set into action by an expiratory current of air from the lungs, very much like the vibrations of an elastic tongue in a reed instrument. In order that the vocal cords may be set in vibration, they must be put in a state of tension and the aperture of the glottis narrowed to afford resistance to the air current. During speech, the cords are brought together by the sphincteric laryngeal girdle. The muscles particularly concerned in this action are the thyroarytenoideus, the lateral cricoarytenoideus and interarytenoideus, all of which, by synergic contraction, approximate the arytenoid cartilages and vocal cords. During phonation, the cords are closed and an expiratory effort is made by which the pressure of air in the trachea is raised. By virtue of the contraction of the thyroarytenoideus muscles, a certain degree of elasticity is given to the cords. The elasticity varies with the strength and contraction of these muscles. The elasticity of the glottic margins causes the vocal cords to come in apposition during the lowering of pressure, but immediately as they do so the pressure again rises and the cords are blown apart. This escape of puffs of air takes place in a rhythmical manner and thus a musical sound is produced.

Aside from the laryngeal mechanism as described above, the lungs, cavities of the pharynx, mouth and nose contribute

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to normal voice production. The lungs develop the necessary air pressure, and the cavities of the pharynx, mouth and nose modify the intensity of the tone and the relative intensity of the overtones. These mechanisms (the larynx, lungs and pharynx) are entirely dependent on nervous control, the principal part of which is played by the VIIth, Xth and XIIth nerves, and partially by the XIth. These cranial nerves act as intermediaries to the higher centres of the brain.

It is thus not difficult to appreciate that, aside from the local disturbances in the larynx (tumors, ulcers, inflammatory swellings, etc.), there can be innumerable causes for disturbances of the voice by any dissociation of the neurogenic hookup from brain to larynx proper, or to other regions that are factors in voice production. Diseases of the upper and lower respiratory tract, systemic diseases and diseases of the surroundings of the larynx may be responsible for disturbances in the voice. The causes of such changes are numerous and should be known not alone to the laryngologist but to the general practitioner, who must be made to appreciate the importance of voice changes. A complete classification of the causes for hoarseness follows:

1. Inflammatory (nonspecific): *a.* acute laryngitis; *b.* chronic laryngitis; *c.* abscess of larynx; *d.* perichondritis; *e.* membranous laryngitis (nondiphth.); *f.* fibrinous corditis; *g.* acute infectious laryngotracheitis.

2. Inflammatory (specific) (laryngeal complications in specific infectious diseases): *a.* Tuberculosis; *b.* syphilis; *c.* diphtheria; *d.* Vincent's ulceromembranous laryngitis; *e.* influenza; *f.* measles; *g.* scarlet fever; *h.* blastomycosis and other fungi; *i.* typhoid fever; *j.* anthrax; *k.* leprosy; *l.* smallpox; *m.* rhinoscleroma; *n.* trichinosis; *o.* glanders; *p.* rabies; *q.* typhus.

3. Trauma: *a.* External trauma with fracture of larynx; *b.* burns from radium and caustics; *c.* misuse of voice with cord hemorrhage; *d.* foreign body in larynx; *e.* dislocations of larynx; *f.* stab wounds and gunshot wounds.

4. Anomalies: *a.* Laryngoptosis; *b.* acromegaly; *c.* congenital webs or cysts; *d.* ventricular prolapse; *e.* laryngocele; *f.* lateral cervical fistula; *g.* double vocal cords.

5. Allergy: a. Angioneurotic edema; *b.* serum disease; *c.* urticaria.

6. Metabolic — Circulatory and Blood Diseases: a. Passive congestion in chronic nephritis, cardiac failure and cirrhosis of liver; *b.* anemia; *c.* agranulocytosis; *d.* leukemias; *e.* diabetes; *f.* myxedema; *g.* gout.

7. Associated with Skin Disorders: a. Pemphigus; *b.* herpes; *c.* lupus; *d.* scleroderma; *e.* erysipelas; *f.* impetigo; *g.* xanthoma; *h.* lichen ruber planus.

8. Occupational Diseases (industrial dusts and chemical inhalations): a. Stone, metal and wood; *b.* sulphuric, nitric and picric acids; *c.* gases in modern warfare; *d.* intense heat and steam (cooks, stokers and firemen).

9. Benign Tumors: a. Angioma, hematoma, fibroma, polyp, varix, teratoma, lipoma, chondroma, papilloma, lymphangioma, cysts, pachyderma, keratosis and nonspecific granuloma.

10. Malignant Tumors: a. Carcinoma and sarcoma.

11. Neurogenic: A. Central Lesions: a. Bulbar paralysis; *b.* tumors of pons and medulla; *c.* disseminated sclerosis; *d.* syringomyelia; *e.* tabes; *f.* meningitis; *g.* tetanus; *h.* strychnine poisoning; *i.* aneurysms of basilar and vertebral artery; *j.* medullary syndromes involving nucleus ambiguous (Avellis, Tapia, Schmidt, Vernet, Jackson, Collet and Secard).

B. Peripheral (pressure or injury to recurrent or vagus nerve): a. Enlarged or substernal thyroid; *b.* after thyroid surgery; *c.* tumors in neck, trachea or esophagus; *d.* aneurysm of aorta or subclavian artery; *e.* mediastinal glands or tumors; *f.* pleural adhesions; *g.* apical tuberculosis; *h.* scoliosis of cervical vertebrae; *i.* enlarged heart or pericardial effusions; *j.* syndrome of jugular foramen (tumors or caries at base of skull or jugular thrombophlebitis).

C. Peripheral Neuritis (recurrent laryngeal): a. Alcohol; *b.* lead; *c.* tobacco; *d.* arsenic.

D. Other Neurogenic Causes: a. Spasm of larynx; *b.* laryngismus stridulous; *c.* tremor of larynx; *d.* myasthenia laryngis; *e.* hysterical aphonia.

12. Miscellaneous: a. Cricoarytenoid arthritis; *b.* contact ulcer; *c.* general feebleness; *d.* puberty changes; e. aphthous ulcer; *f.* drugs (potassium iodide, opium, belladonna and pilocarpine).

From the above classification it is evident that hoarseness is a symptom present in a great variety of diseases, and the laryngologist need be mindful of these many pathologic entities. Frequently impairment of the voice may be the first symptom of a disease remote from the larynx but secondarily affecting this organ. The alert throat specialist, aware of the many possibilities responsible for hoarseness, is thus often able to be a great help to the internist in arriving at a diagnosis. For example, the patient who presents himself to the throat specialist with the complaint of hoarseness and whose larynx on examination reveals an ulcerated or infiltrated process will be quickly advised to have a blood Wassermann, a complete blood count and an X-ray of chest taken. In such a manner, pulmonary tuberculosis, syphilis or a blood dyscrasia may be detected. Again, a patient with hoarseness, on examination, may show a paresis of one or both vocal cords. Such a finding may lead to the diagnosis of an aneurysm of the aorta, an enlarged heart, a mass in the mediastinum or a neurological entity, such as tumors of pons or medulla, disseminated sclerosis, syndrome of the jugular foramen and others. Examination of the larynx may also reveal a massive symmetrical edema which suggests the possibility of an allergic state. These are some of the interesting phases concerned with hoarseness. To illustrate specifically, I briefly report the following unusual cases that had been under my care, and which demonstrate how intensely interesting a case of hoarseness may be, and how our diagnostic acumen may be taxed to the utmost before arriving at a conclusive opinion.

Case 1: F. A., male, age 21 years, employed in a fur establishment, presented himself to me for examination on Oct. 21, 1936, with a complaint of hoarseness for a period of nine weeks. Examination of the larynx revealed an immobile left cord and arytenoid. The left cord was in midline position. The right cord and arytenoid moved normally. No tumefaction, ulceration or inflammatory changes were evident in the larynx. The pyriform sinuses were clear. A diagnosis of left recurrent paralysis was made and its etiology undetermined. Laboratory tests were then suggested, and the following are reports of these examinations.

X-ray of chest, basal metabolism and Wassermann were done on Oct. 31, 1936. They were negative. Blood examination for arsenic on Nov. 27, 1936, after an arsenic-free diet, was reported as 0.025 mg. per 100 cc. of blood. Blood examination for lead on Dec. 17, 1936, was reported

as 0.16 mg. per 100 cc. of blood. Complete blood count on Dec. 4, 1936, showed R.B.C., 4,030,000; W.B.C., 6,300; hemoglobin, 75 per cent; differential: neutrophiles, 70; lymphocytes, 29, and eosinophiles, 1 per cent.

In view of the presence of arsenic and lead as reported, and for absence of any other positive findings to account for the recurrent paralysis, a diagnosis of mild lead and arsenic poisoning was made, with a peripheral neuritis involving the left recurrent laryngeal nerve. There was no other symptom or sign of lead or arsenic poisoning. The patient was placed on a diet, and when seen on June 9, 1937, had a very good voice. The left cord had definite motion but still was sluggish.

Thus, this case represents a very unusual occurrence of peripheral neuritis involving the recurrent laryngeal nerve due to lead and arsenic.

Case 2: A female, age 69 years, had been admitted to the Post-Graduate Hospital on the service of Dr. Charles J. Imperatori[1] on Feb. 4, 1936. Her symptoms were hoarseness, loss of weight and vomiting. She had been previously hospitalized at another hospital, where she had been esophagoscoped, and a report of this examination indicated that a constriction was found at the lower part of the esophagus. Laryngeal examination at the Post-Graduate Hospital revealed a left cord in the cadaveric position and much mucoid secretion in left pyriform sinus. X-ray of chest showed an aortic heart and bronchiectasis. Examination of esophagus with barium was negative. Five days after admission, the patient developed a bronchopneumonia and she died on Feb. 16, 1936. In view of her pneumonia, an esophagoscopy had not been performed. An antemortem diagnosis was not made. A postmortem examination revealed a periaortic fibrosis with inclusion of left vagus nerve which caused the left cord paralysis. Also, autopsy demonstrated general arteriosclerosis, bronchiectasis and bronchopneumonia. The esophagus was not found diseased. The periaortic fibrosis probably had resulted from fibrosis of glands in this region.

The hoarseness in this case is easily explained by the inclusion of the recurrent laryngeal fibres in the fibrosis.

Case 3: M. F., negress, female, age 49 years, was admitted to Riverside Hospital on July 30, 1937. Examination revealed an extensive bilateral tuberculosis with submiliary involvement on the right side, and extensive pyopneumothorax on the left. She gave a history of being hoarse all of her life; otherwise there was nothing else pertinent in her past personal history. Her sputum was positive and her Kahn was negative. A routine examination of the larynx was attempted during her confinement in the hospital but she was very unco-operative, making it impossible to visualize her larynx. In view of her very toxic condition, no further attempt was made to examine her. Her course was steadily downhill and she died on Nov. 22, 1937. An antemortem description of her larynx was thus not obtainable. In view of the presence of active tuberculosis, a presumptive diagnosis of tuberculous laryngitis was made.

Anatomical diagnosis at autopsy was extensive bilateral tuberculosis with cavitation and left pyopneumothorax; tuberculous enterocolitis; generalized amyloidosis. The larynx did not reveal any edema, swelling or ulceration. The right ventricular band and vocal cord were normal. The left ventricular band was normal. The left vocal cord was somewhat thinner than the right and was reduplicated somewhat beneath itself by another similar cord running parallel to the first, merged with it at its extreme ends and separated from it over its entire length by a narrow but rather deep cleft — a double vocal cord.

This case[2] of a double vocal cord is a very unusual one. Only five other such cases have been reported heretofore, all in the foreign literature.

To arrive at a diagnosis of a patient presenting the symptom of hoarseness is of the utmost importance. The classification of the various diseases that are responsible for this symptom have been given above, and the means of reaching an absolute diagnosis will be, to a great extent, by careful elimination of all possibilities. A patient with hoarseness requires a thorough examination, to include a complete history and physical examination, laryngoscopy, indirect or direct, when the mirror examination is not satisfactory, an X-ray of chest and occasionally of the neck, Wassermann, complete blood count and urine analysis. Biopsy should be performed when there is the slightest suspicion of a malignancy. Bronchoscopy and esophagoscopy need occasionally be performed.

One need bear in mind that a patient with hoarseness, whose chest has been X-rayed and declared to be positive for pulmonary tuberculosis, need not always have a laryngeal tuberculosis. Of course, one is quickly prejudiced with such a finding, but it must be borne in mind that a chronic laryngitis, a malignancy or syphilis may be responsible for such hoarseness. Similarly, a 4+ Wassermann in a patient with hoarseness does not preclude that the laryngeal pathology is luetic. The occurrence of tuberculosis and syphilis in the same larynx is not rare, and the coexistence of tuberculosis, syphilis and malignancy in the larynx has been reported.

When a specimen of tissue is taken for a histological examination in a case suspicious of malignancy, one must realize that a negative report need not always be conclusive. It is not unusual to find a biopsy specimen reported as negative, and, when repeated, showed malignant changes. With the knowledge that malignant changes take place in the deep cells, it can readily be seen that when a bit of tissue is taken that has not penetrated the depths of the new growth, the pathological report will not reveal malignant changes; however, when a lesion does look suspicious, it is well to repeat the biopsy one or more times before excluding the possibility.

A typical case demonstrating the presence of a malignancy in a patient with a 4+ Wassermann which had been treated as a luetic laryngitis is herewith given. The case also emphasizes the need for repeated biopsies.

Case 4: H. C., age 45 years, presented himself at the Stuyvesant Polyclinic Dispensary on Oct. 28, 1936, with a history of hoarseness for a

period of seven months. Examination of larynx revealed a markedly swollen right ventricular band which prevented visualization of the right cord. The left ventricular band was ulcerated, as was the left vocal cord. There was much interarytenoid thickening. An X-ray of chest and a Wassermann were immediately taken. The chest plate showed no evidence of tuberculosis and the Wassermann was reported as 4+. The larynx appeared to be inflammatory, and in view of a positive Wassermann, a diagnosis of luetic laryngitis was made. Antiluetic treatment was instituted. After five weeks of intensive treatment, the larynx, rather than showing evidence of improvement, was more extensively involved. This prompted me to take a biopsy on Dec. 4, 1936. The pathological report was chronic inflammatory tissue. Antiluetic treatment was continued and again with no evident improvement. A second biopsy was taken on Jan. 8, 1937, and also was reported as chronic inflammatory tissue. The local pathology in the larynx continued to progress, and the glottic edema was becoming more and more pronounced. On several occasions I had advised hospitalization for a tracheotomy, but not until Jan. 20, 1937, did the patient consent to this operation. The tracheotomy was performed on Jan. 22, 1937, at the Post-Graduate Hospital, service of Dr. C. J. Imperatori. With the tube in situ, and with little fear now of reaction edema to biopsy, I did a third biopsy. The pathological report was epidermoid carcinoma, Grade II. Not appearing to be a good surgical risk, the patient was referred for deep X-ray therapy. He received radiation from March 9, 1937, to May 27, 1937. The immediate response seemed very favorable but later there had been extensive glandular metastases as well as progressive advancement of the laryngeal disease. He died on April 16, 1938.

CONCLUSIONS.

I have presented herewith an original classification for the causes of hoarseness. I have also briefly reported four cases of hoarseness to demonstrate the unusual causes for this symptom and the difficulties often encountered in arriving at a diagnosis. At times this may be a very simple matter, as it would be in the identification of a localized, benign or malignant growth, but at other times the determination of a diagnosis may become a most difficult and painstaking procedure.

The relation between the laryngologist and the internist is also demonstrated.

The method of approach for a diagnosis of any complaint referable to the larynx has been outlined.

The need for repeated biopsies has been stressed in a case suspected of being malignant.

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OTORHINOLOGIC SEQUELAE OF SWIMMING. ANALYSIS OF PRESENT CONCEPTS. A NEW METHOD OF PREVENTION.

DR. DAVID MEZZ, Brooklyn.

There is no national sport today that can claim as many active participants as swimming. Several forces have contributed to this truly phenomenal growth in popularity:

1. There is the diligence of the health agencies, such as our Committee on Swimming Hygiene, which is appointed by the Section of Laryngology, Otology and Rhinology of the American Medical Association; such as the engineering divisions of our Federal and State health services; and as the various public health associations, all of which have contributed immeasurably to the sanitation and safety in and about pools and beaches.

2. There is the general recognition that swimming has the dual capacity of providing both wholesome mental relaxation and superb physical training. In fact, it is the ideal exercise because it calls into play the general body musculature and produces the elongated, smoothly functioning type of muscle in a uniformly developed body.

3. The strongest force, which has brought countless numbers into the ranks, which has stimulated a race upon the part of municipalities and schools everywhere to construct pools, and which has made swimming compulsory for the average student and camper throughout the country, is the influence of such well informed organizations as the American Red Cross, the Young Men's and Women's Christian Associations, the College and High School Swimming Coaches Associations, the Boy Scouts, Girl Scouts and other nationally accepted groups.

These authoritative bodies are responsible in good part for our nation of swimmers by reason of their persistent, well directed efforts to improve the sport and to promote swim-

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ming for exercise, for pleasure, for competitive values and for personal safety.

Coincident with this development, there has appeared augmenting evidence which points to the etiological rôle of swimming, and especially diving, in the production of otorhinological infections. From the articles in the medical periodicals and from a good many most informative letters addressed to the author by leading rhinologists in all parts of the country, it becomes apparent that aquatic activity is universally regarded to be a prominent etiological factor in the incidence of sinusitis and otitis media.

It is not the purpose of this paper to present a detailed study of the subject in all its phases. Rather is it meant to review the literature briefly, to bring under experimental scrutiny some fallacies in present day concepts and, finally, to offer the profession both a new viewpoint and a simple solution of the problem, based on several years of practical testing.

REVIEW OF THE LITERATURE.

The standard textbooks contain surprisingly little information on the subject. There is a brief reference in "Jackson and Coates" which states that "otitis media may occur from accidental forcing of water into the ear while swimming." One other, in "Logan-Turner," reads tersely, "bathing and diving have been recorded as etiological factors in sinusitis."

In contrast to this paucity of information, there is considerable literature which has appeared in the journals, commencing with 1921, when Fenton[6] reported a case of bilateral thrombosis of the lateral sinuses in a patient with an antecedent history of much swimming and diving. This patient developed an acute suppurative pansinusitis, which was quickly complicated by orbital and intracranial involvements.

Fenton[8] again, in 1922, reported three additional cases, in each of which bathing was followed by a severe and complicated sinusitis. This paper cited similar reports by Cobb, in 1908; Wilkinson, in 1912, and Hope, in 1914, the latter two having presented a number of cases before the Royal Society of Medicine. It is interesting to note that Fenton recom-

mended the prophylactic use of heavy mentholated oil in the nose prior to swimming activity, and that Dr. Ross Hall Skillern in his discussion on the paper advised the plugging of the nose with absorbent cotton in order to obviate the entrance of water into the nasal spaces.

Much important information has been contributed by Taylor. In the first of a series of papers, this author[9, 10] noted the frequency of otorhinologic difficulties following swimming, and brought forth the predisposing factors of faulty, crowded noses, infected sinuses and prolonged chilling of the body surfaces. Subsequently, Taylor[12] elaborated on the subject of man's lack of adaptation to aquatic habits by presenting a scholarly study of the comparative anatomy which indicates why "man is susceptible to sinus disease while lower animals, breathing air as man does and living constantly in the water do not encounter these difficulties." Referring to his own investigations and to those of Wiedersheim,[2] Eckley[1] and Reese,[5] Taylor showed that diving mammals are provided with a complicated, sphincter-like musculature which shuts the nostrils tight during submersion of the head. So endowed are the alligator, the beaver, the hippopotamus, the walrus, the seal and the menatee. Also, the diving birds having external nostrils, like the diving petrel, have musculature arrangements similar to the alligator whereby their nostrils may be closed very tightly. Further, those submerging birds having no external nares, as the booby, the cormorant and the snakebird, possess a small elongated tongue which can be plugged directly and tightly into the internal nares. Nature has endowed these creatures with muscular structures whereby they are able to isolate their nasal mucosa from the contact with water.

By contrast, man possesses a small decadent muscle, which is rarely demonstrable in the dissecting room and which is known as the compressor narium. Unfortunately, its functional ineffectuality is only heightened by its phylogenetic significance, for here we have the simple answer to this particular phase of the problem. Man cannot close his nostrils reflexly or voluntarily to protect his nasal interior under similar conditions.

Taylor[12] continued with the second of the anatomical differences between man and other mammals. The latter possess

a thick layer of nonconducting blubber; some have a heavy coat of fur in addition. These structures insulate the mammal from the loss of body heat. The human swimmer, lacking this insulation, is unable to withstand over-exposure to cold, as proven by the author in an experiment involving 250 school children, in whom body temperature was reduced several degrees after a period of 45 minutes in water of 73° F. Trommsdorf[3] agrees with this view and adds that prolonged chilling of the body causes a decreased motility and phagocytic power of the leukocytes.

In 1926, the annual report of the Committee on Otorhinologic Hygiene of Swimming contained pertinent advice warning persons with colds, head infections and running noses or ears, that swimming tends to force infections into the sinuses or ears, which might result in serious if not fatal consequences. Diving feet first was condemned, and bathers were further urged not to extend their swimming sessions beyond 45 minutes.

It remained for Hasty[14] to devise a test whereby he proved that "water in the pools during the time of swimming represents the combined washings, so to speak, of the nasal mucus membranes of every swimmer." Sterile powdered charcoal was thrown into a pool and a class of boys was allowed to take the usual swimming exercises. At the end of the period, an inspection of the nasal chambers was made. In every boy, particles of the charcoal were found about the middle turbinate, which led Hasty to observe that "water in the majority of swimmers gets well into the nasal chambers, carrying with it the contamination of the pool and the swimmers' own noses, and at the same time adding to the pool whatever bacteria may be present in the particular swimmer's nose. The infections are likely transmitted from one individual to another during the swimming period before the sterilization process has time to influence the bacterial count materially." This statement coincides with the recent opinion of Skillern,[47] who wrote that "one swimmer with a sinus infection can easily pollute pool water, which may be taken into the nostrils of his fellows in a concentrated form." Hasty concluded that "otorhinologic infections caused by swimming have become so frequent in recent years that almost every family has

in one way or another been brought to grief as a result of swimming."

Fenton[20] and his committee of 1928 warned swimmers who employed the various styles of immersion swimming now in vogue that nasal exhalation is imperative whenever the face is under water. Again, emphasis was placed on the washing of the patient's own germs into the unprotected sinus and ear zones by the forcible inrush of water or by the patient's efforts at removal. In an individual paper, Fenton[21] elaborated on these statements and strongly condemned feet-first diving. The writer reiterated the importance of exhaling through the nose and inhaling through the mouth. A short study was included on the effect of plain water on turbinate tissue in which there was observed distinct evidence of imbibition, especially in the submucosal layer; that is, swelling of the cell bodies and poor staining of the nuclei. Fenton then stated that salt water is less destructive than plain water in its effects on nasal mucosa.

Stark[22] experimented with rabbit nasal mucosa which he irrigated with water. He noted varying degrees of change, both macroscopically and microscopically. There was a hyperemia with marked leukocytic infiltration, increased glandular activity and increase in the size of the vacuoles and epithelial cells.

Again, Taylor,[29] in 1932, presented an excellent motion picture study which demonstrated the anatomical advantages of the diving mammals who possess muscular sphincters to close their nostrils during submersion. Additional emphasis was placed on the danger of feet-first diving, an opinion which concurs with the recent newspaper reports of complaints being made by our American divers who are objecting strenuously to a change in the Olympic diving rules, wherein they are compelled to execute several feet-first entries. The coach of this group has pointed out the incidence of sinus and ear infections to his stellar performers through such forced activity.

While the literature received no contribution in the two years that followed, yet the 1934 report of the Committee on Otorhinologic Hygiene of Swimming stated that "numerous reports during the past year of fulminating types of frontal sinusitis with an accompanying osteomyelitis,

attributed to swimming and diving, cannot but stimulate the otorhinologist."

Possessing an excellent background, both as an American Red Cross swimming instructor and afterwards as an otolaryngologist, Saunders[24] advocated the use of a clamp to close the nostrils for feet-first divers. From his intimate knowledge of the technique of swimming, Saunders analyzed the various styles of swimming and stated that the splash over the head in the backstroke makes it practically impossible to keep water out of the nose. A new method of exhalation was recommended; that is, through the nose and mouth simultaneously. In many styles of swimming on the front or the side, the face is carried beneath the surface a good part of the time, owing to the necessity for holding the head in line with the spine in order to get better body position and balance in the stroke. Thus, for example, the good crawl stroke swimmer actually does not lift his nostrils out of the water throughout his swim. Certainly, nasal exhalation cannot continue throughout this immersion, which makes the advice to keep exhaling through the submerged nose a matter of superfluity.

Taylor and Dyrenforth,[42] in a very recent study, repeated their investigation of the factor of body surface chilling, from which they concluded that chilling without exercise produces a leukopenia of the polymorphonuclear neutrophilic type, thus impairing the phagocytic powers of the fixed tissue cells, including that of the nasal mucosa.

Summarizing the salient information which was gathered in this review of the literature, we learn that:

1. There is universal agreement on the rôle which swimming and diving play in the etiology of sinusitis and otitis media.

2. The infections so incurred are frequently more severe and attended by complications in a greater percentage of cases.

3. Our normal endonasal defense mechanism consists of columnar ciliated epithelium covered by a thin film of mucin, which is inhibitory to bacterial growth and in which the bacteria are enmeshed. This mucus film moves towards the pos-

terior choanae in a steady flow, carrying entangled bacteria and foreign matter to the nasopharynx, to be swallowed or expectorated. The motive power for this mechanism is supplied by the columnar ciliated epithelium, the cilia of which wave sufficiently strong, according to Parkinson[39] to move test droplets of ink at the rate of 0.5 cm. per minute, or enough to move the mucus from anterior to posterior nasal limits within 10 minutes. It is stated that the most important single factor in the prevention and eradication of nasal infection is the preservation of this mucus film and the maintenance of its usual movement by normal ciliary activity.

4. The endonasal invasion of water disrupts this normal defense mechanism in one or more ways, as follows:

a. The mucus film is washed away.

b. The ciliary activity is impaired, either by the thermal shock or by the chemicals contained in the water.

c. Micro-organisms are washed from the highly immunized lower airways to the feebly resisting sinuses or Eustachian tubes.

d. There may occur direct mechanical trauma to ostea and to mucosal linings by the forcible inrush of water, especially if chlorinated, which produces first an ischemia and then a reactive swelling, a combination which permits water to enter the sinuses easily and then subsequently blocks its exit. Especially is this true in the congenitally narrow nose, the nose with septal or turbinate deformities, and the nose with excessive adenoidal growths.

Mahoney's descriptive remark[45] can summarize these changes when he says: "I have never seen a professional swimmer whose nasal mucosa did not resemble the appearance of a dishwasher's hands."

5. Microscopically, there occurs the manifest evidence of reaction to insult; hyperemia, leukocytic infiltration, engorged cavernous spaces, hyperactive mucus glands, swollen cells and poorly staining nuclei. As Taylor stated it, "anything that interferes with the normal defense mechanism, whether it is mechanical, chemical or thermal, breaks down Nature's bar-

riers and renders the nasal cavity and its appurtenances as susceptible to infection."

6. A number of predisposing factors encourage these infections; such as the overloaded pool with too many bathers or too inadequate a circulating system, which results in a high bacterial content; such as over-chlorinated pools, which are irritating and prepare a receptive groundwork for the implantation of pathogenic organisms in the nasal mucosa; as the bathers with colds or active head infections, those with low resistance, or those remaining overlong in the water. On this last factor, Furstenberg[18] comments that 30 minutes is safe bathing time, saying, "I mention this time limit because there is a concession on the part of most writers that man can resist the loss of body heat in water at about 60° F. for a period of approximately 30 minutes."

EXPERIMENTAL STUDIES.

As stated early in this paper, the writer questioned several statements which are repeated in the literature. One was the importance of body chilling as the all-inclusive cause of the otorhinologic sequelae of swimming. A brief limited test was made, as follows:

EXPERIMENT I—CHILLING OF BODY SURFACES.

Procedure: Two guinea pigs were placed in a refrigerator for 50 minutes. At the end of this period, one was killed immediately. The second was killed after cold pool water was introduced into its nose. The turbinate tissue of both animals was sectioned and revealed the interesting information as noted in the microphotographs on the following page:

Discussion: As in the experimental findings of Fenton[21] and Stark,[22] the writer observed the reactive microscopic changes when the nasal mucosa was bathed by chlorinated water, regardless of the factor of body chilling. There is no attempt to minimize the importance of this latter factor. It is the author's opinion, however, that entirely too much clinical evidence supports these microscopic findings to relegate the factor of "direct water damage" to a position of secondary importance, as has been done by the disciples of "body chilling." Thus, we all have witnessed the raw, irritated feeling

of the nasal interior, the immediate stuffiness, the quickly produced sense of discomfort, pressure or fullness in the head or ears, the dripping of water from the nose, the squirt of

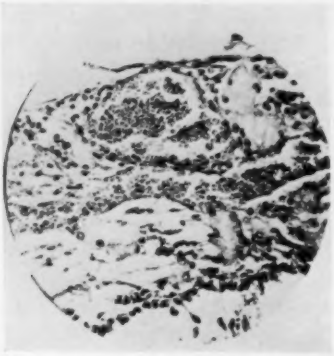

Fig. 1. Chilling alone. Normal mucosa with little demonstrable change. Normal structure of columnar ciliated epithelium, beneath which is a loose connective tissue stroma, in which thin wall cavernous spaces are noted.

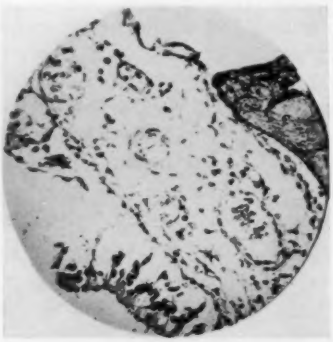

Fig. 2. Chilling plus water invasion. Intense congestion, cavernous spaces are engorged, leukocytic infiltration, hyperactive mucus glands.

water upon bending forward; the rushing, bubbling or thunderous sound in the ears, followed by a period of decreased hearing, the jogging on one foot, the acute ache or pressure above an eye or in a cheek. All these are signs of water

invasion which disrupts, *per se*, the normal defense mechanism and physical condition of the nasal interior.

Perhaps the confidence which these investigators have had in the capacity of nasal exhalation to protect the swimmer from the invasion of water into the nasal interior has been responsible for the focusing of their attention on the factor of body chilling. This brings the writer to the second series of experiments, which has the purpose of testing the efficacy of respiration as a protective force.

It is not commonly known that there is a confusion of advice in the highest and most authoritative swimming circles exactly through which channel the air should be exhaled. The old stand-by was "exhale through your nose," or, through the nose and mouth simultaneously. Recently, the American Red Cross, the body which is generally accepted as the leading authority on swimming matters, came forth in their official publication[44] with the emphatic advice that the nose be excluded and the mouth be used for exhalation as well as inhalation.

The writer, whose sinusitis came as a consequence of aquatic activity and who is intimately acquainted with the rules of breathing and the various styles of swimming questioned the protective value of any respiratory maneuvers to exclude water from the nasal chambers. In this view, he is joined by a great many of the important swimming people with whom he has been in personal communication and who express their disbelief at the notion that swimmers can keep water out of their noses. With this background, there followed a series of experiments to test the techniques and effects of breathing:

EXPERIMENT II – STATUS OF RESPIRATION DURING DIVING.

Procedure: Six well trained divers were instructed to fill their lungs with a deep inhalation, do a front jackknife dive and exhale slowly while submerged, in accordance with the usual instructions. As each diver's head reappeared at the surface he exhaled into a respirometer whatever air there remained in his lungs and the readings in cubic centimetres were charted. Again, the divers were instructed to take a deep inspiration, do a front jackknife dive but, now, to suspend all

breathing from the moment they left the diving board until they emerged and expelled all the remaining air into the respirometer. The results are charted below:

TABLE I.
Measurements of Expired Air After Breaking Water.

Subject	Exhalation (Continued Throughout Under-Water Arc) Measured in Cubic Centimetres by Respirometer	Exhalation (Suspended Throughout Under-Water Arc) Measured in Cubic Centimetres by Respirometer
J. L.	1,400 cc.	1,500 cc.
G. S.	1,250 cc.	1,200 cc.
W. M.	1,350 cc.	1,400 cc.
A. S.	1,400 cc.	1,300 cc.
L. U.	900 cc.	950 cc.
T. S.	1,600 cc.	1,500 cc.

Discussion: The writer is convinced that during a dive, the tidal and complementary airs are lost in the effort of the dive, respiration is suspended — a natural reflex response to the excursion through space — and the diver completes his under-water arc with nothing more in his lungs than the supplementary air remaining of his original vital capacity. Reduced to simple terms, we really do no breathing during the dive, and pierce the water with surprisingly little air remaining in the lungs. As stated concisely in the detailed report of Prof. Chaikelis,[44] "the amount of air expired by the diver at the end of the dive appears to be definitely less than the measured supplemental air. The explanation as to this rather interesting phenomenon, in view of the observation that the diver generally takes what is called a 'deep breath,' is that during the muscular effort exerted in the performance of the dive, there results an escape of air through nose and mouth, in spite of the voluntary attempt to hold the breath. If we accept this view, that air is lost from the respiratory tree during the act of diving, we can see then why no water tends to enter the nasal passages in the initial penetration-of-the-water phase of the actual dive. Apparently a sufficient 'positive pressure' of air is created to prevent water from entering the nasal passages; however, it is a fact that water does penetrate into the nasal passages and sinuses during the under-water phase and the breaking-water phase of the dive,

the last-mentioned phase being responsible for the introduction of most of the water. Whatever air is retained in the respiratory tree, which may act as a wall, is compressed into a small space of nasopharynx, and with some of it even being forced into the Eustachian tubes and being responsible for the tenseness of the eardrums. Thus, the water tends to displace the air because not enough air pressure exists to prevent this exchange taking place."

EXPERIMENT III – SIMULTANEOUS NOSE AND MOUTH EXHALATION.

Procedure and Discussion: A good many smokers were asked to expel their cigarette smoke simultaneously through mouth and nose. This group included a well known instructor and disciple of this combined form of exhalation. In no case was there a successful attempt, and the explanation is apparent. When the mouth is used for exhalation, the soft palate is kept high and posteriorly. When the nose is used for exhalation, the palate is placed in the low and anterior position. In order to master simultaneous mouth and nose exhalation, one would be compelled to re-educate the reflexes of the soft palate so that it remains in a midposition. At best, this takes on the status of a stunt which, even when mastered by the few, accomplishes no apparent purpose.

EXPERIMENT IV – POSTURE AND BREATHING.

Procedure: The six well trained swimmers were put through studied exercises in a chlorinated pool over a period of days. After each session, absorbent cotton was placed in the middle meatus and permitted to remain there while the subjects donned their clothing. The cotton was then removed and placed in a test tube, to which was added distilled water. The tube was shaken vigorously in order to incorporate the contents of the cotton into the distilled water. The cotton was squeezed thoroughly and then discarded. Now, 10 drops of 25 per cent silver nitrate were added to each test tube and a rough evaluation of the resulting white precipitate of silver chloride was charted as follows:

TABLE II.

Qualitative Analysis of the Amount of Chlorine in the Middle Meatus as Determined by the Silver Nitrate Test.

	J. L.	G. S.	W. M.	A. S.	L. U.	T. S.
Quiet face-down float with nasal exhalation	0	0	0	1+	0	0
Quiet face-down float with mouth exhalation	0	0	0	0	0	1+
American crawl with nasal exhalation	2+	1+	1+	1+	0	3+
American crawl with mouth exhalation	1+	1+	0	2+	0	2+
Breast stroke with nasal exhalation	2+	1+	2+	2+	0	3+
Breast stroke with mouth exhalation	3+	1+	1+	1+	2+	3+
Back stroke with nasal exhalation	3+	2+	2+	3+	2+	3+
Back stroke with mouth exhalation	3+	3+	2+	3+	1+	3+
Single jackknife dive	2+	2+	2+	3+	3+	2+
Feet-first dive	3+	4+	3+	4+	4+	3+
Six jackknife dives	3+	4+	4+	4+	3+	4+
Single dive and five-minute swim	2+	2+	2+	3+	3+	3+

Discussion: A study of this chart elicits some very interesting observations, as follows:

a. Quiet face-down floating is most effective in preventing the ingress of water.

b. Neither nasal nor mouth exhalation excludes water from the nose.

c. The worst offenders are feet-first diving, repeated diving and backstroke swimming.

These conclusions were rechecked by means of a glass nose, designed by the writer, the interior of which was shaped to simulate the turbinate bones and meati. Attaching a short rubber tube of good sized calibre, clamped at the free end, created the equivalent of the positive nasal pressure to which the literature refers. By moving the glass nose through the water in the various positions of diving and swimming, the author obtained a visual verification of the previous findings. Again, the feet-first divers, the repeat divers and the backstrokers piled up the greatest quantity of water in our glass nose.

These findings were essentially substantiated in another and more significant way. Two of our volunteers had stuffy noses and a watery drip for several hours, and one other complained of a full head and an ache above the left eye following the diving sessions. We noticed that J. L. hopped around after most of the tests, trying to dislodge the water that had found

its way into the middle ear by way of the Eustachian tube. Here again, we have the clinical evidence of water invasion — the nasal twang to the voice, the "raw feel" in the nose, the stuffiness, the full head, the localized ache, the impaired hearing, the jarring of the head, the dripping of water, the noises in the ears — these are the indicators that present day academic concepts on exhalation as a protecting force are fallacious.

Nature did not anticipate terrestrial man's rebirth of interest in a water existence and so discarded the all-important

Figs. 1 and 2.

muscle, the compressor narium, which would have solved this phase of the problem of otorhinologic sequelae by a natural function, as in the case of air-breathing mammals and birds who live and dive in the water with impunity.

The final phase of this paper deals with the development of an artificial substitute for our lost compressing muscle. This idea was not original with the author, as previously mentioned. A great variety of methods have been used to block off the nostrils. The heavy oil, the stuffing of the nose with cotton, the strapping of the nares with adhesive tape, the use of all sorts of clothes pins and clamps — all this indicates the continued interest upon the part of afflicted or interested

individuals to eliminate the troublesome ingress of water. It might be pertinent to report here that the latest publication of the American Red Cross, in discussing sinus infection and diving, states, "to prevent water penetration, all that is necessary is to keep the mouth closed and pinch the nostrils shut with a clip device."

For the past two years the writer has been testing the efficacy of a rubberized, spring-steel nose clip (See Figs. 1, 2, 3 and 4), which is constructed to conform to the anatomical

Fig. 3.

planes of the external nose. Critical study of its efficiency in preventing post-swimming discomfort has brought him to the following conclusion: when worn correctly and used diligently, this rubberized nose clip supplies a satisfactory substitute for our decadent compressor narium. Users of this device have been significantly free of nasal disturbances despite protracted sessions and stunting in the water. This device is now formally presented to the profession as the simple and logical answer to the problem of sinus and ear infections consequent to aquatic indulgence.

The author wishes to express his unqualified thanks to Mr. William Mullen, Director of Swimming at Erasmus Hall High School, for his immeasurable and continuous assistance in the practical testing; his gratitude to Prof. Chaikelis, of the Department of Biology, the College of the City of New York, for his invaluable assistance in the physiological studies, and

to Dr. Leo Meyer, of the Pathology Department, the Wyckoff Heights Hospital, for his co-operation in the animal and tissue investigations.

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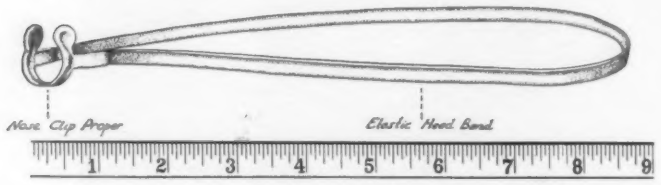

Fig. 4.

Marshall Taylor, Jacksonville; Dr. T. B. Wood and Dr. H. Meyersburg, Brooklyn, and Dr. A. C. Furstenberg, Ann Arbor.

To the many swimming coaches and their students who co-operated so earnestly in the practical application and testing of the nose clip in the past two years, the writer can only express his sincere thanks. It has been a source of constant encouragement to have received their enthusiastic approval and to have afforded them healthful indulgence in this sport.

SUMMARY.

1. Swimming and diving are potent factors in the etiology of otorhinologic diseases.

2. Diving animals are protected by muscles which close the nostrils, and by a coat of insulation which protects them from the vitiating effects of loss of body heat.

3. Lacking these anatomical features, man is exposed to the invasion of water into the nasal anterior and to the reduction of body heat.

4. The concept that nasal exhalation, mouth exhalation or combined exhalation is adequate to hold back the water is proven to be fallacious.

5. The limit of bathing time should be 45 minutes.

6. A nose clip is recommended for swimmers and divers, especially those who tend to catch cold easily, those having a sinus or ear history, those with anatomically or pathologically crowded noses, and those who are professionally engaged in considerable aquatic activity.

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NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Meeting of Nov. 15, 1939.

(a)—Late Complications of Brain Abscess, with Case Presentations. Dr. Ira Cohen.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(b)—Brain Abscess, with Case Presentations. Dr. Abraham Kaplan (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(c)—Management of Brain Abscess of Otorhinogenic Origin. Dr. Joseph E. J. King.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. FOSTER KENNEDY: At the beginning of this discussion tonight, accepted principles were spoken of; Dr. King's work has overturned accepted surgical principles in this domain.

When I began to know something — not much — about brain abscesses, all my seniors looked upon brain abscess as almost the most fatal of all sicknesses of the brain. It really wasn't until Dr. King produced his idea of open surgery, openly arrived at, that we began to find different results. Speaking as a physician, I cannot but feel that all blind surgery, whether it be the pushing of hollow instruments into a brain abscess that you don't see, or the pushing of 80 per cent alcohol into a nerve that only the patient feels, or the pushing of alcohol into even paravertebral ganglia, is either bad surgery or lucky surgery. Surgery, to be effective, must be directed surely by the senses of the operator, and the best sense is that of sight; and the second best is that of touch.

So Dr. King refused to tap and tap and tap until death took away the material for tapping; he sliced the top off the abscess and looked in. Having established the first principle right, his technique could hardly fail to get the right result. That is not so just in medicine, it is so throughout everything. Ideas are tops. Techniques are second tops. Good technique can make a bad principle seem to work until it blows up in your face, but good principles — sound principles — can, if once established, enable almost a duffer — not a complete duffer, but almost a duffer — to make them come out right.

Dr. King has got completely right principles here for dealing with brain abscesses. Again and again, I have seen his ideas work, and again and again before that I have seen the other idea of repeated taps fail; so that I am glad and happy to be here to corroborate by word of mouth what he has been saying to you.

Dr. King spoke of the periabscess — edema around the abscess; an important idea, that, and one that sometimes gives great difficulty in diagnosis. Sometimes there is no abscess. Sometimes there is an adjacent toxic focus with an edema highly localized, producing an alteration of function which gives the appearance of abscess. I don't mean at all to go into the question of diagnosis; the subject of tonight's discussion is the management of brain

abscess. But I would be false to my own trade if I did not say that without correct diagnosis, management fails. There is nothing much to management if you will but follow the King technique. You must know, first, that there is an abscess, and where there is an abscess, before you can manage an abscess.

There is a tendency which has become rather a cliché, rather a routine thing, to give too much water to your man just because he is sick. "Pressing fluids" is almost a routine remark thrown over the shoulder to the nurse as the physician or surgeon leaves the ward. But "pressing fluids" in cases of brain disease sometimes kills the patient by disturbing water balance, giving rise to medullary edema, respiratory failure and death. It is important to know when not to press fluids — for edema of the brain kills as many patients as do tumors and abscesses.

Notice that I said a word about principles being half of ideas, but notice, too, Dr. King's technique of getting a piece of gauze out of a brain by bubbling it out with hydrogen peroxide instead of abstracting it with dissecting forceps. The requisite agent is applied and the time is given to raise the gauze out of the brain without injury. This technique is not sufficiently understood in high places.

There are surgeons who insist on following what they call their own ideas in the management of brain abscesses. I believe I have had a considerable experience with brain abscess, and a considerable observation experience of their management, and I have not the least hesitation in saying that there is no way of managing a brain abscess like this one. This is the *only* way in which such abscesses should be managed.

Dr. King failed to tell you, I think, that somewhere back in his pedigree he must have been sired by a St. Bernard dog! For after he has taken the top off an abscess, after he has seen the abscess, after he has given it authority and power to herniate itself — he comes down every night and lies under the bed, and dresses the wound.

Dr. Isidore Friesner: I mean to be very brief, because I feel that this discussion should be delivered over to those who are neurologists and neurosurgeons. Naturally, I have nothing to offer constructively in the care of these patients. Now, those of you who with me attended otologic sessions 20 or 25 years ago or more heard that statement made repeatedly by otologic surgeons, and, as a matter of fact, the reason for that, I believe, was that the experience of any one of us was so limited in regard to brain abscess that we did not have a sufficient amount of material from which we could really learn very much. Twenty or 25 years ago or more, I was led to the belief that these cases offer the type of problem whose solution is much better attempted, let me say, by the neurosurgeon. It is true that in the beginning I was prodded a little so that this conclusion was not entirely spontaneous, but I did not take the attitude that I have described in regard to these cases until I was convinced that the neurosurgeon was correct. Since that time I have handled no brain abscesses and, therefore, naturally I have nothing constructive to offer in the way of their therapy.

I am very happy, too, with this decision for two reasons: I need only to call to your attention the material that has been shown to you this evening to make you realize that the efforts of the neurosurgeon are attended by far greater success than those of the otologist, at least in the old days. I said I was very happy for two reasons — first, because I am convinced that the patients with brain abscess have a far better chance in the hands of the neurosurgeon than they have in the hands of the otologist; and secondly, I think I have the edge on Joe King a little bit on this, because I have an opportunity to catch up on my fishing, and Joe has to continue to take care of brain abscesses.

Dr. Robert E. Buckley: I would like, first of all to express my sincere compliments to Dr. King and Drs. Cohen and Kaplan for what so obviously

represents years of hard clinical study, and for their ability to express to us, in a few short minutes, a comprehensive concentrate of that labor.

The role assigned to me as rhinologist is rather difficult, as brain abscess as the result of sinusitis is a rather rare complication. I was stunned yesterday when in telephoning to Dr. King he assured me that I must be wrong in this idea, because he quickly and without hesitation told me that frontal lobe abscess as the result of sinusitis is second only to brain abscess of ear origin. I felt rather chagrined at my ignorance until, after considerable thought, I realized that almost every brain abscess is the result of a bad ear or a bad nose, and, consequently, Dr. King was correct in his statement that the nose ranks second as a causative factor. He forgot to tell me that the ratio was about one sinus abscess to about 100 ear abscesses.

Meningitis, and not brain abscess, is the usual fear of the rhinologist. Clinically, it has been my experience that the average brain abscess is accidentally found by the rhinologist during an external sinus operation, at which time, after the usual incision, a necrotic or missing posterior sinus wall is found with an ulcerating mass leading directly to a brain abscess. Most of these cases have no symptoms to indicate brain involvement. This may be explained by the fact that the frontal lobe is relatively silent, consequently giving no localizing symptoms. Generalized brain symptoms do not appear because increased intracranial pressure is prevented by the absence of any mechanical obstruction. This is obvious, since the abscess is directly connected with the frontal sinus and drains very freely into the nose, so that no mechanical obstruction can exist. The so-called loss of recent memory and personality changes which are attributed to the frontal lobe involvement, I believe, are too rare to be commonly expected.

From a rhinological standpoint of the pathways of infection to the meninges, I feel that they can be divided as follows: First, ethmoiditis normally produces a direct extension through the cribriform plate, practically always producing a meningitis. Second, a cavernous sinus thrombosis results usually in localized, and then a generalized meningitis. Third, chronic frontal sinusitis, with or without a missing posterior wall, is the usual cause of a brain abscess, insofar as the nose is concerned. This abscess is usually located directly behind the posterior wall of the frontal sinus.

I have had the pleasure to see Dr. King operate and, consequently, I haven't any comments to make except that I agree with him entirely in his idea that radical surgery is indicated.

Dr. Lewis D. Stevenson: Your Chairman has asked me to bring up some pathological specimens, and I have brought one or two which perhaps might interest you. I had collected a number of brain abscesses that came to autopsy at Bellevue, with a view to trying to illustrate for you the development of the capsule from early in the illness up to about four and one-half months, but I don't think I have time to illustrate that fully tonight. I shall just confine myself to one or two things that have struck me in studying these specimens, and to showing you a few lantern slides that have a message for us, I think.

Just a word about the pathogenesis of these lesions. I think the pathway of infection, which has been neglected in considering this subject, is the blood vessel itself entering the brain, rather than the perivascular space about the vessels, or retrograde thrombosis of a vein, or arterial thrombosis. I think our sections show many cases in which the infection is confined to the wall of the vessel rather than to the spaces about it.

The other thing I should like to illustrate for you is that abscesses are frequently multiple, as Dr. King and others have pointed out. You may be dealing with a number of abscesses, or one with a number of pockets not easily seen at first. You can see that illustrated in the first specimen, and in a slide or two which I shall show you in a moment. Then, also, I should like to point out that if a hernia is allowed to develop, it may bottle up a sinus

which is purulent and so end in a fatal encephalitis or meningitis. Two instances of that I have here tonight.

I should like to point out, too, that the reaction in the brain to abscess is entirely different from trauma or hemorrhage into the brain. In abscess, the capsule is formed by fibroblasts — there is very little reaction on the part of the neuroglia.

I might say a word about this multiple abscess specimen, about four and one-half months old, as far as we can tell by the clinical history of brain disease. The patient came in with osteomyelitis of the petrous bone and was operated on at least four times, once before the case came to Bellevue. A frontal abscess was located and allowed to come up and herniate, but apparently there were still two cavities behind the hernia, which became quite large, as you see, and which resulted in the death of the patient.

(Slide) Here is the first case of herniation. It was an abscess of the brain, which was treated by cutting open and allowing the brain to herniate; but the hernia has come out and bottled up a sinus which was purulent, finally leading to the death of the patient.

(Slide) Here is an ordinary abscess of the temporal lobe from mastoid disease, with a fairly thick capsule of fibrous tissue of about two and one-half months' duration.

(Slide) Here is the abscess cavity I have just shown you, and here is the thick fibrous tissue wall. In the middle of this several months' development of capsule there is another abscess, quite fresh and causing a reaction in the brain which undoubtedly could give us trouble all over again. You see a great deal of encephalitis about this secondary abscess within the capsule of the main abscess.

(Slide) This is the specimen which is on the table, in which the brain was allowed to herniate, but not directly over these abscesses which were confined within the brain and not evacuated by this method of treatment.

(Slide) Here is a slide from the same case, taken a little further back. The hernia can be seen here, illustrating the fact that if there is attachment to the dura or scalp, the ventricle may be enormously dilated and bulge into the wound.

(Slide) View of the wall of the capsule, with the secondary abscess within the original abscess wall.

If anybody would like to look at them, I have a number of slides here which show the abscess wall stained green by trichome stain.

Dr. Ira Cohen: The Chairman referred to Dr. King's talk before this Society 15 years ago. I recall some five years after that discussing a paper of Dr. King's, and at that time I made the remark that we were not as far apart in our handling of abscess as we seemed to be. As the years have rolled on now, I would say we are not at all apart, but that the treatment of abscesses is viewed through exactly the same eyes. We must agree that the open method is the only one for the adequate handling of abscesses.

It was rather an unfortunate choice, perhaps, to show patients with complications because, of course, they do not represent the operative results we have obtained. But I deliberately picked out those that did not have a smooth postoperative course, and those who as late as five or six years after discharge developed complications.

Dr. Abraham Kaplan: I should like to say that in spite of excellent otologic or rhinologic surgery, a brain abscess may form: and that unless the primary focus is very widely removed, a recurrence of the brain abscess is

very likely. It is that part of the problem which is completely in the hands of the rhinologists or otologists.

Concerning the treatment of brain abscess, there are a great many things which cannot be brought out either in a paper or in a talk, but can only be learned by watching such a master as Dr. King.

Dr. Joseph E. J. King: I want, first, to thank everyone for the kind things they have said, and Dr. Stevenson for his demonstration.

I have learned one thing which I consider important during the observation of six patients, and I want to leave this fact with you tonight. I know that a number of cerebral abscesses have been cured by the use of drainage tubes; however, in some cases in which a drainage tube is used the patient will be improved for four or five days and then relapse, and in the six cases spoken of, the reason for the relapse is as follows: A cross-section of an abscess in the cerebral hemisphere is approximately round or circular. When one puts a tube into the middle of the abscess, the patient gets along well for a while, but after four or five days he may not do so well and may again become comatose. When one removes the dressing and looks at the wound, no pus is seen coming through the tube. Still the patient is quite ill. One may think that all the pus has escaped, but here is what has happened: (Blackboard demonstration showing collapse of circular abscess cavity into a clover-leaf-shaped figure or cavity with three undrained pockets.) With this condition present, one should place the patient on the side and do a lumbar puncture. It will be necessary to remove 30 or 45 cc. of cerebrospinal fluid. The abscess will gradually begin to open up again, and one will see pus dammed back in the three lateral pockets, and it will begin to escape as the cerebrospinal fluid leaks through the lumbar puncture needle. The abscess again becomes a more opened, rounded cavity, so that it can be properly cleaned out, inspected and packed in the usual manner with iodoform gauze. In view of the fact that the lumbar puncture is done on the fourth or fifth day, the meningocortical area will have become well walled off, and there will be no danger of a leakage of pus producing meningitis at this site. The subsequent manner of dealing with the cerebral abscess is as described in the paper.

BOOK REVIEW.

Surgery of Injury and Plastic Repair. By Samuel Fomon. Pp. 1409, illustrations 925. 1939. Williams Wilkins Co., Baltimore. $15.00.

This is a tremendous volume of 20 chapters, ranging from "Fluid, Salt and Acid Balance" through "Cleft Lip and Cleft Palate." This reviewer does not pretend to have read all of the book; to do so would produce a review many months hence. The main idea obtained is of a huge reference list and a condensation of what has been published by other authors, *viz.*: *1.* a total of 2,725 references; *2.* 111 references in the 16-page chapter on shock; *3.* 342 in the 217-page chapter on the nose.

It seems like a yearbook de luxe, and on the cover are the statements: *1.* 'This is a book for every surgeon, every specialist and *every practitioner of medicine*," and *2.* "Surgery that makes the patient's life more livable."

There are many borrowed statements and illustrations, and on first attempt at study, the method of reproduction makes the illustrations seem sort of out of focus. For all the subject matter covered, there are less than a half dozen original "before and after" pictures, and these of hump noses, ears and face lifts. The remaining few illustrations of patients are taken from the Medical Department, United States Army, Volume XI. J. B. B.